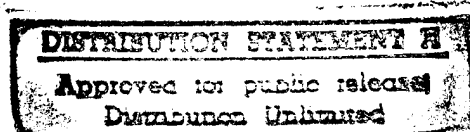


**United States Air Force  
611th Air Support Group/  
Civil Engineering Squadron**

**Elmendorf AFB, Alaska**

**Final  
Risk Assessment**

**Cape Lisburne Radar Installation,  
Alaska**



19960813 173

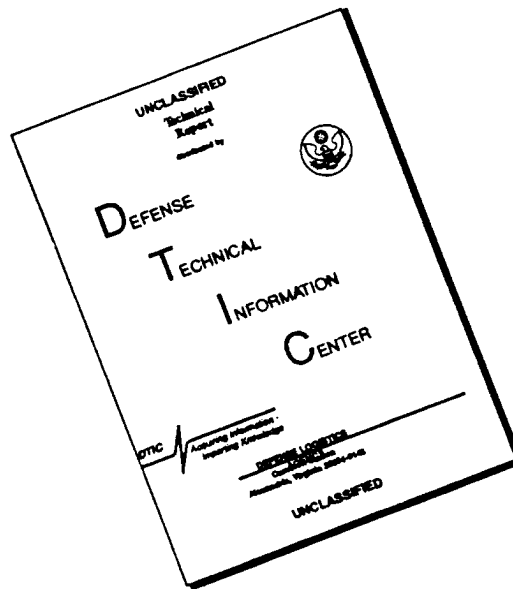
**Prepared by:**

**ICF Technology Incorporated**

**05 FEBRUARY 1996**

THIS QUALITY INSPECTED 1

# DISCLAIMER NOTICE



THIS DOCUMENT IS BEST QUALITY AVAILABLE. THE COPY FURNISHED TO DTIC CONTAINED A SIGNIFICANT NUMBER OF PAGES WHICH DO NOT REPRODUCE LEGIBLY.

## **PREFACE**

This report presents the findings of Risk Assessments at sites located at the Cape Lisburne radar installation in northern Alaska. The sites were characterized based on sampling and analyses conducted during Remedial Investigation activities performed during August and September 1993. This report was prepared by ICF Technology Incorporated.

This report was prepared from July 1995 to February 1996. Mr. Samer Karmi of the Air Force Center for Environmental Excellence Environmental Restoration Division (AFCEE/ESR) was the Alaska Restoration Team Chief for this task. Dr. Jerome Madden and Mr. Richard Borsetti of the 611th CES/CEVR were the Remedial Project Managers for the project.

Approved:

---

Thomas McKinney  
Program Director  
ICF Technology Incorporated

## **NOTICE**

This report has been prepared for the United States Air Force (Air Force) by ICF Technology Incorporated for the purpose of aiding in the implementation of final remedial actions under the Air Force Installation Restoration Program (IRP). As the report relates to actual or possible releases of potentially hazardous substances, its release prior to an Air Force final decision on remedial action may be in the public's interest. The limited objectives of this report and the ongoing nature of the IRP, along with the evolving knowledge of site conditions and chemical effects on the environment and health, must be considered when evaluating this report, since subsequent facts may become known which may make this report premature or inaccurate. Acceptance does not mean that the United States Air Force adopts the conclusions, recommendations or other views expressed herein, which are those of the contractor only and do not necessarily reflect the official position of the United States Air Force.

Government agencies and their contractors registered with the Defense Technical Information Center (DTIC) should direct requests for copies of this report to: DTIC, Cameron Station, Alexandria, Virginia 22304-6145.

Non-Government agencies may purchase copies of this document from: National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, Virginia 22161.



## TABLE OF CONTENTS

1.0	INTRODUCTION .....	1-1
1.1	ORGANIZATION OF REPORT .....	1-1
1.2	RISK ASSESSMENT GUIDANCE DOCUMENTS .....	1-6
1.3	INSTALLATION DESCRIPTION AND ENVIRONMENTAL SETTING .....	1-6
1.3.1	Sites Evaluated at the Cape Lisburne Radar Installation .....	1-12
1.4	APPROACH TO HUMAN HEALTH RISK ASSESSMENT .....	1-13
1.5	APPROACH TO ECOLOGICAL RISK ASSESSMENT .....	1-14
2.0	BASELINE HUMAN HEALTH RISK ASSESSMENT .....	2-1
2.1	IDENTIFICATION OF CHEMICALS OF CONCERN .....	2-1
2.1.1	Sampling Strategy and Evaluation of Analytical Data .....	2-2
2.1.2	Risk-Based Screening Levels .....	2-3
2.1.2.1	Formulae for Calculating RBSLs .....	2-3
2.1.3	Screening of Chemicals by Comparing Maximum Detected Concentrations of Essential Human Nutrients .....	2-5
2.1.4	Concentrations of Organic and Inorganic Constituents in Background Samples .....	2-5
2.1.5	Selection of Chemicals of Concern .....	2-9
2.1.5.1	Landfill and Waste Accumulation Area (LF01) .....	2-16
2.1.5.2	White Alice Site (SS03) .....	2-23
2.1.5.3	Spill/Leak #3 (ST07) .....	2-23
2.1.5.4	Upper Camp Transformer Building (SS08) .....	2-23
2.1.5.5	Lower Camp Transformer Buildings (SS09) .....	2-31
2.1.5.6	Summary of Chemicals of Concern .....	2-31
2.2	EXPOSURE ASSESSMENT .....	2-35
2.2.1	Pathway Analysis .....	2-35
2.2.1.1	Soil and Sediment Ingestion .....	2-35
2.2.1.2	Inhalation .....	2-40
2.2.1.3	Water Ingestion .....	2-40
2.2.1.4	Ground Water .....	2-40
2.2.2	Migration and Fate of Chemicals of Concern .....	2-40
2.2.3	Estimation of Chemical Intake .....	2-42
2.2.3.1	Exposure Point Concentration .....	2-43
2.2.3.2	Exposure Frequency .....	2-43
2.2.3.3	Exposure Duration .....	2-43
2.2.3.4	Averaging Time .....	2-44
2.2.3.5	Ingestion of Locally Produced Meat, Fish, and Vegetation .....	2-44
2.2.3.6	Soil Ingestion Rate .....	2-44
2.2.3.7	Drinking Water Ingestion Rate .....	2-44
2.2.3.8	Dermal Contact with Soil Rate .....	2-45
2.2.3.9	Inhalation Rate .....	2-45
2.2.3.10	Body Weight .....	2-45

## TABLE OF CONTENTS (CONTINUED)

2.2.4	Quantifying Exposure .....	2-45
2.3	TOXICITY ASSESSMENT .....	2-47
2.3.1	Toxicity Assessment for Noncarcinogenic Effects .....	2-47
2.3.1.1	Concept of Threshold .....	2-47
2.3.2	Toxicity Assessment for Carcinogenic Effects .....	2-49
2.3.2.1	Concept of Nonthreshold Effects .....	2-49
2.3.2.2	Assigning a Weight-of-Evidence .....	2-49
2.3.2.3	Generating a Slope Factor .....	2-49
2.3.2.4	Identifying the Appropriate Data Set .....	2-51
2.3.2.5	Extrapolating to Lower Doses .....	2-51
2.3.2.6	Summary of Dose-Response Parameters .....	2-52
2.3.3	Summaries of the Toxicity of the Chemicals of Concern .....	2-52
2.4	RISK CHARACTERIZATION .....	2-52
2.4.1	Quantifying Risks .....	2-53
2.4.1.1	Risks from Individual Substances - Carcinogenic Effects ..	2-53
2.4.1.2	Noncancer Hazards from Individual Substances - Noncancerous Effects .....	2-53
2.4.1.3	Aggregate Risks for Multiple Substances .....	2-54
2.4.2	Site-Specific Risk Characterization .....	2-56
2.4.2.1	Landfill and Waste Accumulation Area (LF01) .....	2-60
2.4.2.2	White Alice Site (SS03) .....	2-61
2.4.2.3	Spill/Leak #3 (ST07) .....	2-61
2.4.2.4	Upper Camp Transformer Building (SS08) .....	2-62
2.4.2.5	Lower Camp Transformer Buildings (SS09) .....	2-62
2.5	RISK CHARACTERIZATION UNCERTAINTY .....	2-62
2.6	RISK ASSESSMENT SUMMARY AND CONCLUSIONS .....	2-65
3.0	ECOLOGICAL RISK ASSESSMENT .....	3-1
3.1	SELECTION OF SITE CONTAMINANTS .....	3-2
3.1.1	Surface Water .....	3-8
3.1.1.1	Organic Compounds .....	3-8
3.1.1.2	Metals .....	3-10
3.1.2	Soils and Sediments .....	3-11
3.1.2.1	Petroleum Hydrocarbons .....	3-11
3.1.2.2	Benzene, Toluene, Ethylbenzene, and Xylenes .....	3-12
3.1.2.3	Volatile Organic Compounds .....	3-13
3.1.2.4	Semivolatile Organic Compounds .....	3-15
3.1.2.5	Polychlorinated Biphenyls .....	3-16
3.1.2.6	Metals .....	3-16
3.2	ECOLOGICAL EXPOSURE ASSESSMENT .....	3-18
3.2.1	Species of the Arctic Coastal Plain .....	3-19
3.2.1.1	Plants .....	3-19
3.2.1.2	Aquatic Organisms .....	3-23
3.2.1.3	Birds .....	3-23

## TABLE OF CONTENTS (CONTINUED)

	3.2.1.4 Mammals .....	3-23
	3.2.1.5 Threatened and Endangered Species .....	3-24
3.2.2	Representative Species .....	3-24
	3.2.2.1 Representative Plants .....	3-25
	3.2.2.2 Representative Aquatic Invertebrates and Fish .....	3-25
	3.2.2.3 Representative Birds .....	3-25
	3.2.2.4 Representative Mammals .....	3-27
	3.2.2.5 Threatened and Endangered Species .....	3-27
3.2.3	Exposure Pathways .....	3-27
3.2.4	Habitat Suitability for Representative Species .....	3-28
3.2.5	Exposure Assessment for Representative Species of Plants .....	3-29
3.2.6	Exposure Assessment for Representative Aquatic Organisms .....	3-29
3.2.7	Exposure Estimates for Representative Bird and Mammal Species .....	3-30
	3.2.7.1 Potential Bioaccumulation of COCs in Representative Species .....	3-30
	3.2.7.2 Estimation of Percent Ingested Onsite .....	3-31
	3.2.7.3 Exposure Assessment for Representative Species of Birds .....	3-33
	3.2.7.4 Exposure Assessment for Representative Species of Mammals .....	3-39
3.3	ECOLOGICAL TOXICITY ASSESSMENT .....	3-40
3.3.1	Petroleum Hydrocarbons .....	3-40
	3.3.1.1 Plants .....	3-43
	3.3.1.2 Aquatic Organisms .....	3-43
	3.3.1.3 Birds .....	3-43
	3.3.1.4 Mammals .....	3-44
3.3.2	Benzene .....	3-44
	3.3.2.1 Plants .....	3-44
	3.3.2.2 Aquatic Organisms .....	3-44
	3.3.2.3 Birds .....	3-44
	3.3.2.4 Mammals .....	3-44
3.3.3	Toluene .....	3-44
	3.3.3.1 Plants .....	3-44
	3.3.3.2 Aquatic Organisms .....	3-44
	3.3.3.3 Birds .....	3-44
	3.3.3.4 Mammals .....	3-45
3.3.4	Xylene .....	3-45
	3.3.4.1 Plants .....	3-45
	3.3.4.2 Aquatic Organisms .....	3-45
	3.3.4.3 Birds .....	3-45
	3.3.4.4 Mammals .....	3-45

## TABLE OF CONTENTS (CONTINUED)

3.3.5	1,4-Dichlorobenzene .....	3-45
3.3.5.1	Plants .....	3-45
3.3.5.2	Aquatic Organisms .....	3-45
3.3.5.3	Birds .....	3-45
3.3.5.4	Mammals .....	3-46
3.3.6	4-Methylphenol .....	3-46
3.3.6.1	Plants .....	3-46
3.3.6.2	Aquatic Organisms .....	3-46
3.3.6.3	Birds .....	3-46
3.3.6.4	Mammals .....	3-46
3.3.7	Polychlorinated Biphenyls .....	3-46
3.3.7.1	Plants .....	3-46
3.3.7.2	Aquatic Organisms .....	3-47
3.3.7.3	Birds .....	3-47
3.3.7.4	Mammals .....	3-47
3.3.8	Iron .....	3-47
3.3.8.1	Plants .....	3-47
3.3.8.2	Aquatic Organisms .....	3-47
3.3.8.3	Birds .....	3-47
3.3.8.4	Mammals .....	3-48
3.3.9	Manganese .....	3-48
3.3.9.1	Plants .....	3-48
3.3.9.2	Aquatic Organisms .....	3-48
3.3.9.3	Birds .....	3-48
3.3.9.4	Mammals .....	3-48
3.3.10	Characterization of Effects .....	3-48
3.3.10.1	Toxicity Reference Values .....	3-49
3.4	RISK CHARACTERIZATION FOR ECOLOGICAL RECEPTORS .....	3-57
3.4.1	Potential Risks to Representative Species of Plants .....	3-58
3.4.2	Potential Risks to Representative Species of Aquatic Organisms ..	3-65
3.4.3	Potential Risks to Representative Species of Birds .....	3-66
3.4.4	Potential Risks to Representative Species of Mammals .....	3-67
3.4.5	Potential Future Risks .....	3-67
3.5	ECOLOGICAL RISK ASSESSMENT UNCERTAINTY ANALYSIS .....	3-68
3.5.1	Environmental Sampling and Analysis .....	3-68
3.5.2	Selection of Chemicals for Evaluation .....	3-70
3.5.3	Selection of Representative Species .....	3-70
3.5.4	Exposure Assessment .....	3-70
3.5.5	Toxicological Data .....	3-71
3.6	SUMMARY OF ECOLOGICAL RISK .....	3-72
3.6.1	Potential Risks to Representative Plants .....	3-72
3.6.2	Potential Risks to Representative Aquatic Species .....	3-72
3.6.3	Potential Risks to Representative Species of Birds and Mammals .	3-73

## TABLE OF CONTENTS (CONTINUED)

4.0 REFERENCES .....	4-1
----------------------	-----

### APPENDICES

A	RISK CHARACTERIZATION SPREADSHEETS
B	TOXICITY PROFILES
C	CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS
D	ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS
E	BIOCONCENTRATION FACTOR CALCULATIONS FOR ORGANIC CHEMICALS
F	SCALING FACTOR CALCULATIONS
G	DATA SUMMARY TABLES

## LIST OF TABLES

1-1.	SITES EVALUATED AT THE CAPE LISBURNE DEW LINE INSTALLATION . . . . .	1-5
2-1.	IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT CAPE LISBURNE . . . . .	2-10
2-2.	CHEMICALS WITHOUT RBSLS AND ARARS OBSERVED IN THE SOIL, SEDIMENT, OR SURFACE WATER AT THE CAPE LISBURNE INSTALLATION . . .	2-15
2-3.	SUMMARY OF THE CHEMICALS OF CONCERN AT CAPE LISBURNE . . . . .	2-17
2-4.	EXPOSURE PATHWAY ANALYSIS FOR CAPE LISBURNE HUMAN HEALTH RISK ASSESSMENT . . . . .	2-39
2-5.	EXPOSURE ASSUMPTIONS FOR ESTIMATING CHEMICAL INTAKE . . . . .	2-42
2-6.	EQUATIONS USED FOR ESTIMATING POTENTIAL DOSE . . . . .	2-46
2-7.	TOXICITY CRITERIA FOR NONCANCER EFFECTS OF THE CHEMICALS OF CONCERN FOR CAPE LISBURNE . . . . .	2-48
2-8.	TOXICITY VALUES FOR THE CARCINOGENICITY OF THE CHEMICALS OF CONCERN AT CAPE LISBURNE . . . . .	2-50
2-9.	EPA WEIGHT-OF-EVIDENCE CLASSIFICATION SYSTEM FOR CARCINOGENICITY . . . . .	2-51
2-10.	SUMMARY OF NONCANCER HAZARD AND EXCESS LIFETIME CANCER RISK FOR CAPE LISBURNE . . . . .	2-58
2-11.	SUMMARY OF SITES WITH CONTAMINATION THAT EXCEEDS REGULATORY BENCHMARKS . . . . .	2-66
3-1.	SUMMARY OF CHEMICALS OF CONCERN: SURFACE WATER . . . . .	3-3
3-2.	SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS . . . . .	3-5
3-3.	CHEMICAL/PHYSICAL PROPERTIES . . . . .	3-14
3-4.	REPRESENTATIVE AND SENSITIVE SPECIES AT AIR FORCE ARCTIC RADAR INSTALLATION SITES . . . . .	3-26
3-5.	BIOCONCENTRATION FACTORS FOR SELECTED ORGANIC COMPOUNDS IN WATER . . . . .	3-32
3-6.	LIFE HISTORY INFORMATION FOR THE LAPLAND LONGSPUR, <i>Calcarius</i> <i>lapponicus</i> . . . . .	3-34
3-7.	LIFE HISTORY INFORMATION FOR THE BRANT, <i>Branta bernicla</i> . . . . .	3-34
3-8.	LIFE HISTORY INFORMATION FOR THE GLAUCOUS GULL <i>Larus hyperboreus</i> .	3-35
3-9.	LIFE HISTORY INFORMATION FOR THE PECTORAL SANDPIPER <i>Calidris</i> <i>melanotos</i> . . . . .	3-35
3-10.	SOIL INGESTION ESTIMATES FOR REPRESENTATIVE BIRD SPECIES . . . . .	3-36
3-11.	LIFE HISTORY INFORMATION FOR THE BROWN LEMMING <i>Lemmus</i> <i>trimucronatus</i> . . . . .	3-36
3-12.	LIFE HISTORY INFORMATION FOR THE ARCTIC FOX, <i>Alopex lagopus</i> . . . . .	3-37
3-13.	LIFE HISTORY INFORMATION FOR THE BARREN-GROUND CARIBOU <i>Rangifer</i> <i>tarandus</i> . . . . .	3-38
3-14.	SOIL INGESTION ESTIMATES FOR REPRESENTATIVE MAMMAL SPECIES . . .	3-41
3-15.	CHEMICAL CLASSES OF GRPH AND DRPH . . . . .	3-42

## LIST OF TABLES (CONTINUED)

3-16.	TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE CAPE LISBURNE INSTALLATION . . . . .	3-50
3-17.	TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE CAPE LISBURNE INSTALLATION . . . . .	3-51
3-18.	TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE CAPE LISBURNE INSTALLATION . . . . .	3-52
3-19.	TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE CAPE LISBURNE INSTALLATION . . . . .	3-54
3-20.	COMPARISON OF COC CONCENTRATIONS TO TOXICITY INFORMATION FOR PLANTS . . . . .	3-59
3-21.	RISK CHARACTERIZATION OF REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE CAPE LISBURNE INSTALLATION . . . . .	3-59
3-22.	RELATIVE TOXICITY RANKINGS FOR COMPARISON OF MAMMALIAN AND AVIAN TOXICITY . . . . .	3-60
3-23.	HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE CAPE LISBURNE INSTALLATION . . . . .	3-61
3-24.	SUMMARY OF ECOLOGICAL RISK ESTIMATES AT THE CAPE LISBURNE RADAR INSTALLATION SITES . . . . .	3-73

## LIST OF FIGURES

1-1.	GENERAL LOCATION MAP .....	1-3
1-2.	AREA LOCATION MAP .....	1-7
1-3.	INSTALLATION SITE PLAN .....	1-9
2-1.	BACKGROUND (BKGD) SAMPLE LOCATIONS AND ORGANIC ANALYTICAL RESULTS .....	2-7
2-2.	LANDFILL AND WASTE ACCUMULATION AREA (LF01) SAMPLE LOCATIONS AND ANALYTICAL RESULTS .....	2-19
2-3.	GRAVEL COVERED AREA #1 AT THE LANDFILL AND WASTE ACCUMULATION AREA (LF01) SAMPLE LOCATIONS AND ANALYTICAL RESULTS .....	2-21
2-4.	WHITE ALICE SITE (SS03) SAMPLE LOCATIONS AND ANALYTICAL RESULTS ..	2-25
2-5.	SPILL/LEAK #3 (ST07) SAMPLE LOCATIONS AND ANALYTICAL RESULTS .....	2-27
2-6.	UPPER CAMP TRANSFORMER BUILDING (SS08) SAMPLE LOCATIONS AND ANALYTICAL RESULTS .....	2-29
2-7.	LOWER CAMP TRANSFORMER BUILDING (SS09) SAMPLE LOCATIONS AND ANALYTICAL RESULTS .....	2-33
2-8.	HUMAN HEALTH RISK ASSESSMENT POTENTIAL EXPOSURE PATHWAYS ....	2-37
3-1.	ECOLOGICAL RISK ASSESSMENT POTENTIAL EXPOSURE PATHWAYS .....	3-21



## LIST OF ACRONYMS AND ABBREVIATIONS

ADD	Average Daily Dose
ADEC	Alaska Department of Environmental Conservation
Air Force	United States Air Force
AMNWR	Alaska Maritime National Wildlife Refuge
API	American Petroleum Institute
ARAR	Applicable or Relevant and Appropriate Requirements
AWQC	Ambient Water Quality Criterion
BCF	Bioconcentration Factors
BTEX	Benzene, Toluene, Ethylbenzene, and Xylene
CDI	Chronic Daily Intake
COC	Chemical of Concern
DEW	Distant Early Warning
DRPH	Diesel Range Petroleum Hydrocarbons
ECAO	Environmental Criterion Assessment Office
EPA	U.S. Environmental Protection Agency
ERA	Ecological Risk Assessment
GRPH	Gasoline Range Petroleum Hydrocarbons
ha	Hectares
HEAST	Health Effects Assessment Summary Tables
HQ	Hazard Quotient
HSDB	Hazardous Substance Data Bank
HVOC	Halogenated Volatile Organic Compound
IRIS	Integrated Risk Information System
IRP	Installation Restoration Program

## LIST OF ACRONYMS AND ABBREVIATIONS (CONTINUED)

IS	Onsite Dietary Intake
LADD	Lifetime Average Daily Dose
LOAEL	Lowest-Observed Adverse Effect Level
LOEL	Lowest Observed Effect Level
MDEP	Massachusetts Department of Environmental Protection
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
PAH	Polycyclic Aromatic Hydrocarbons
PCB	Polychlorinated Biphenyl
RBSL	Risk-Based Screening Level
RfD	Reference Dose
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
RI	Remedial Investigation
RRPH	Residual Range Petroleum Hydrocarbons
SF	Slope Factor
SIF	Scaling Factor
SVOC	Semivolatile Organic Compound
TPH	Total Petroleum Hydrocarbon
TRV	Toxicity Reference Value
TSCA	Toxic Substances Control Act
UCL	Upper Confidence Limit
UF	Uncertainty factors

## **LIST OF ACRONYMS AND ABBREVIATIONS (CONTINUED)**

USFWS	U.S. Fish and Wildlife Service
UST	Underground Storage Tank
VOC	Volatile Organic Compound
WOE	Weight-Of-Evidence

THIS PAGE INTENTIONALLY LEFT BLANK

## 1.0 INTRODUCTION

This document contains the baseline human health risk assessment and the ecological risk assessment (ERA) for the Cape Lisburne radar installation. Five sites at the Cape Lisburne radar installation underwent remedial investigations (RIs) during the summer of 1993. The presence of chemical contamination in the soil, sediment, and surface water at the installation was evaluated and reported in the Cape Lisburne Remedial Investigation/Feasibility Study (RI/FS) (U.S. Air Force 1996). The analytical data reported in the RI/FS form the basis for the human health and ecological risk assessments. The primary chemicals of concern (COCs) at the five sites are diesel, gasoline, solvents, and polychlorinated biphenyls from past spills, leaks, and/or past waste disposal practices. The general location of the Cape Lisburne radar installation is shown in Figure 1-1. The five sites investigated and the types of samples collected at each site are presented in Table 1-1.

The purpose of the risk assessment is to evaluate the human and ecological health risks that may be associated with chemicals released to the environment at the five sites investigated during the RIs. The risk assessment characterizes the probability that measured concentrations of hazardous chemical substances will cause adverse effects in humans or the environment in the absence of remediation. The risk assessment will be used to determine if remediation (site cleanup) is necessary and, if so, to rank sites for remedial action.

### 1.1 ORGANIZATION OF REPORT

Section 1.0 contains introductory information regarding the installation location and conditions, and a summary outline of the approach to the human health and ecological risk assessments. Section 2.0 is the Baseline Human Health Risk Assessment and Section 3.0 is the Ecological Risk Assessment. References are presented in Section 4.0. Section 2.0, Baseline Human Health Risk Assessment, is composed of:

- **Selection of Site Contaminants.** Presents the COCs for human health and describes how they were selected for this risk assessment.
- **Exposure Assessment.** Identifies the pathways by which potential human exposures could occur, and estimates the magnitude, frequency, and duration of those exposures.
- **Toxicity Assessment.** Summarizes the toxicity of the selected COCs and the relationship between magnitude of exposure and the development of adverse health effects.
- **Risk Characterization.** Integrates the toxicity and exposure assessments to estimate the potential risks to human health from exposure to chemicals in environmental media.

THIS PAGE INTENTIONALLY LEFT BLANK

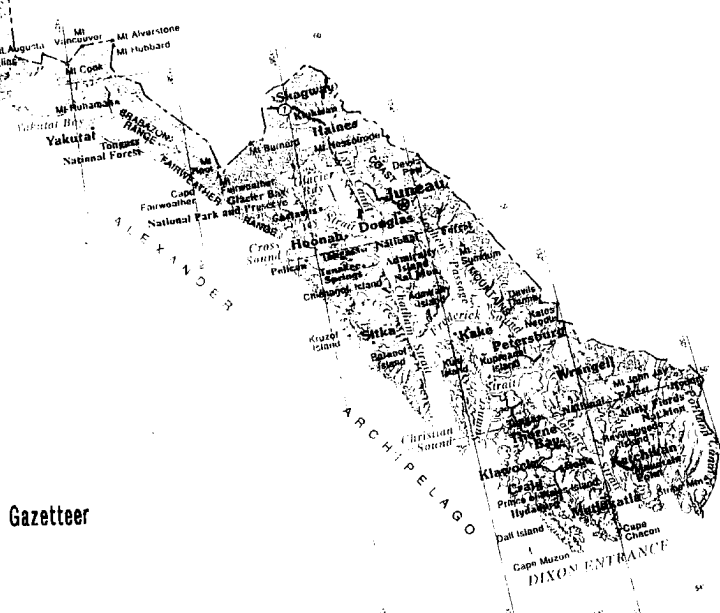


**▲ RADAR SITE**

### GENERAL LOCATION MAP



Source: Alaska Atlas & Gazetteer





**TABLE 1-1. SITES EVALUATED AT THE CAPE LISBURNE DEW LINE INSTALLATION**

SITE NAME	SITE ID NUMBER	SOIL	SEDIMENT	SURFACE WATER
Landfill and Waste Accumulation Area	LF01	X	X	X
White Alice Site	SS03	X	NA	NA
Spill/Leak #3	ST07	X	X	X
Upper Camp Transformer Building	SS08	X	NA	NA
Lower Camp Transformer Building	SS09	X	NA	NA

X Chemical analyses were performed on these media.  
 NA No chemical analysis was performed.

- **Risk Characterization Uncertainty.** Describes the potential shortcomings in the data and the methods used to develop the risk assessment, and the uncertainties in the interpretation of the data and the risk characterization results.
- **Risk Assessment Summary and Conclusions.** Presents summaries and conclusions of the potential human health risk associated with exposure to contaminated media at the five sites at the Cape Lisburne radar installation.

Section 3.0, the Ecological Risk Assessment, is composed of:

- **Selection of Site Contaminants.** Presents the COCs for ecological receptors and describes how they were selected for the ERA.
- **Ecological Exposure Assessment.** Identifies the potential receptors and representative species, habitat suitability, and exposure pathways.
- **Ecological Toxicity Assessment.** Describes the potential effects of site contaminants on the representative species.
- **Risk Characterization for Ecological Receptors.** Evaluates the likelihood of adverse effects on ecological receptors.
- **Ecological Uncertainty Analysis.** Describes the potential shortcomings in the data and the methods used to develop the ERA, and the uncertainties in the interpretation of the data and the ecological risk characterization results.
- **Summary of Ecological Risk.** Presents a summary of ecological risks associated with contaminated media at the five sites at the installation.

Appendix A contains the human health risk assessment spreadsheets used to estimate chemical intake, noncancer hazard, and excess lifetime cancer risk. Appendix B consists of toxicology profiles. The exposure calculations and equations for ecological receptors are presented in Appendices C through F. Appendix G contains a summary of RI sampling and analysis and the RI analytical data for all sites from which the COCs were selected and upon which the human health and ecological risk assessments are based.

## 1.2 RISK ASSESSMENT GUIDANCE DOCUMENTS

The following guidance documents were used to develop the human health and ecological risk assessments:

- *Risk Assessment Guidance for Superfund: Volume 1, Human Health Evaluation Manual (Part A)* [U.S. Environmental Protection Agency (EPA) 1989a];
- Region 10 Supplemental Risk Assessment Guidance for Superfund (EPA 1991a);
- *Risk Assessment Guidance for Superfund: Volume 2, Environmental Evaluation Manual* (EPA 1989b);
- General Guidance for Ecological Risk Assessment at United States Air Force (Air Force) Bases (MITRE 1990);
- Handbook to Support the Installation Restoration Program (IRP) Statements of Work (U.S. Air Force 1991); and
- Framework for Ecological Risk Assessment (EPA 1992a).

## 1.3 INSTALLATION DESCRIPTION AND ENVIRONMENTAL SETTING

The Cape Lisburne radar installation is located at 68°52'N, 166°15'W in the western portion of the Arctic Coastal Plain, along the shore of the Chukchi Sea, and within the Alaska Maritime National Wildlife Refuge (AMNWR). The installation was constructed in 1952 and 1953, occupies 1,125 acres, and consists of two camps connected by a 3.9-mile road. Radar equipment is located at the Upper Camp, and facilities for the support of the radar equipment are located at a Lower Camp. Facilities at the Lower Camp include living quarters, garage, warehouses, POL tanks, water tanks, and a 5,009-foot gravel runway. Several buildings in the Lower and Upper Camps are currently inactive and have not been used since the 1970s when over 100 personnel were stationed at the installation. An area location map is presented in Figure 1-2, and a site plan of the installation is shown in Figure 1-3.

**Geology and Geography.** Geologically the Lower Camp area consists of surficial deposits up to 50 feet thick, comprised of highly permeable sand, silt, gravel, cobbles, and large boulders.

DRAWING No. LIS-AREA

Cape  
Lisburne

Cape Lisburne  
Radar Site



Legend

Approximate property  
boundary

Scale 1:63,360

**CAPE LISBURNE  
RADAR INSTALLATION**

**USAF 611th CES**

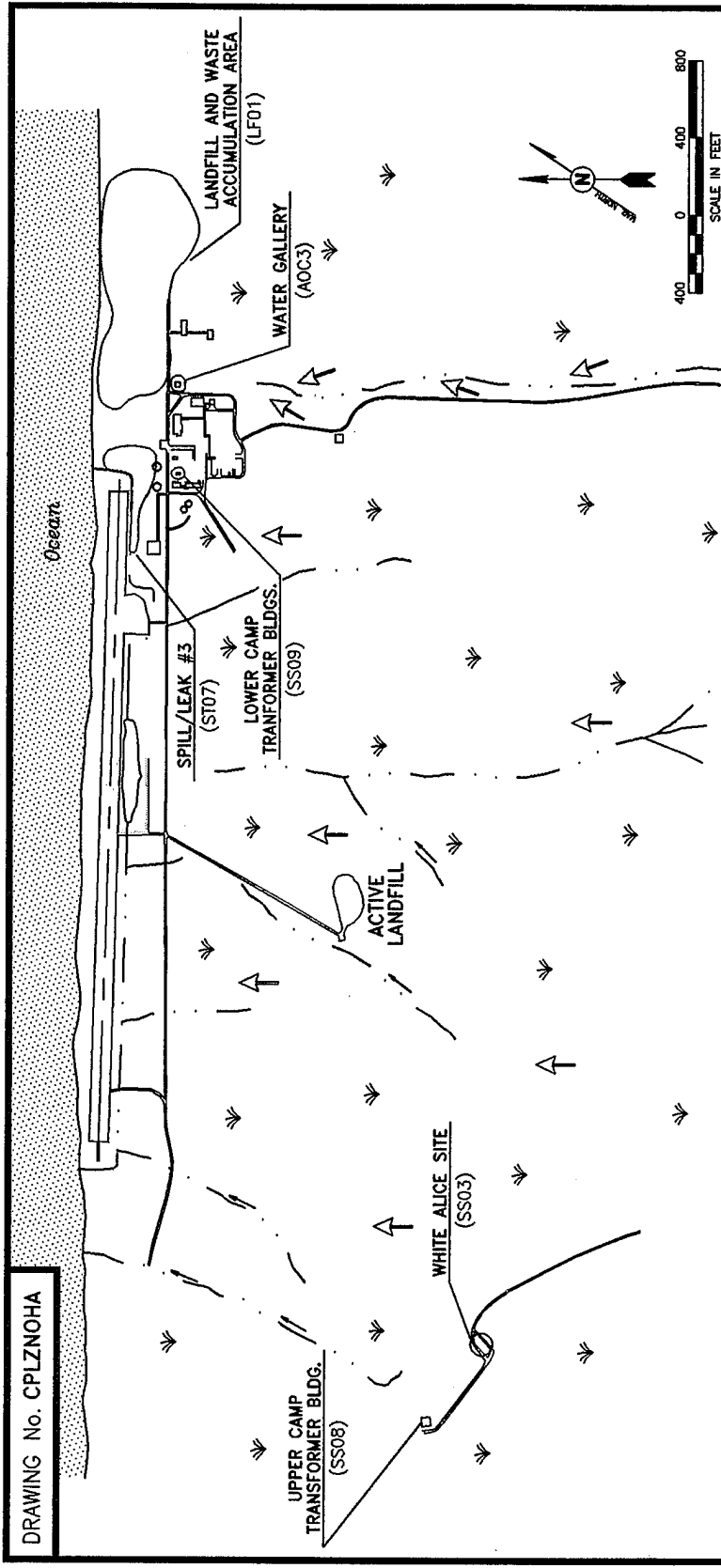
FIGURE NO. 1-2

AREA  
LOCATION MAP

SOURCES: USGS 1952  
USAF 1974 (Updated 1991)

THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. CPLZNOHA



# CAPE LISBURNE RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 1-3  
INSTALLATION  
SITE PLAN

## LEGEND

- BUILDINGS, STRUCTURES
- ROADS
- TUNDRA
- SURFACE WATER
- RIVER, STREAM, OR CREEK
- SURFACE DRAINAGE
- RI SITES AND THE AREA OF CONCERN

THIS PAGE INTENTIONALLY LEFT BLANK

The Upper Camp geology consists of a thin, gravelly layer overlying bedrock at shallow depths and is typical of steeper slopes in the area. Permafrost underlies the entire installation.

Permafrost comprises geologic, hydrologic, and meteorologic characteristics that result in permanently frozen ground. Permafrost occurs in both unconsolidated sediments and bedrock. Its distribution is continuous on the Arctic Coastal Plain, and it has a significant impact on the flow of ground and surface water.

Permafrost acts as an impermeable barrier to the movement of groundwater because pore spaces are ice-filled in the zone of saturation. Recharge and discharge are, therefore, limited to unfrozen channels penetrating the permafrost zone. Permafrost restricts the downward percolation of water and increases runoff, enhancing the creation of lakes and swamps, and also restricts an aquifer's storage capacity and the number of locations from which groundwater may be withdrawn.

The interval between permafrost and ground surface is called the active zone because it freezes and thaws with seasonal weather changes. Water may be found in the active zone in the summer months. The thickness of the active zone at Cape Lisburne varies from approximately one to six feet.

**Meteorology.** Temperatures at Cape Lisburne are generally low throughout the year with a mean annual temperature of 18°F. Annual precipitation at the installation averages 12.3 inches, and prevailing winds are from the east at an average speed of 10 mph. Air quality is generally excellent due to the persistent winds and absence of major air pollution sources.

**Demographics.** Approximately four contract personnel are stationed at the Cape Lisburne installation. The nearest settlement is Point Hope located 35 miles to the southwest with nearly 700 residents, of whom more than 90 percent are Inupiat (Harcharek 1994). There is no road that connects Point Hope and the Cape Lisburne installation.

Of the employed individuals in Point Hope, 93 percent work for the federal, state, or North Slope Borough governments. Approximately 36 percent of the households in Point Hope derive half or more of their meals from subsistence activities based on an average of 21 meals per household per week (Harcharek 1994).

The Cape Lisburne radar installation was investigated to evaluate the possible contamination related to Air Force activities and historical waste disposal practices at the sites. Five sites at the Cape Lisburne installation were determined to be of potential concern based on previous IRP sampling activities, literature review, pre-survey and reconnaissance trips, interviews of station personnel, and information on disposal practices at Distant Early Warning (DEW) Line stations. The sites were investigated during RI/FS activities to confirm the presence or absence of chemical contamination; define the extent and magnitude of confirmed chemical releases; gather adequate data to determine the magnitude of potential risks to human health and the environment; and gather adequate data to identify and select the appropriate remedial actions for sites at which these risks exceed acceptable limits. The remainder of this section describes the five sites where sampling and chemical analyses were conducted during RI activities.

### 1.3.1 Sites Evaluated at the Cape Lisburne Radar Installation

Five sites at the installation that were investigated in the RI were evaluated in the risk assessment. Figures of the five sites are presented in Section 2.0. These sites include:

- **Landfill and Waste Accumulation Area (LF01)** - This site consists of three contiguous areas east of the main installation. The site is covered by gravel on the east and tundra on the west. Two small gravel areas are located in the middle of the tundra-covered section adjacent to the road (gravel covered areas #1 and #2). The site was reportedly used for the disposal of waste oils, paints, solvents and diesel fuels, empty drums, discarded vehicles, and scrap metal. In 1977 to 1978 a general cleanup was performed that included burial of empty drums and other debris, and off-site shipment of drums containing liquid wastes. RI sampling and analyses showed the presence of volatile organic compounds (VOCs), benzene, toluene, ethylbenzene, and xylene (BTEX), gasoline range petroleum hydrocarbons (GRPH), diesel range petroleum hydrocarbons (DRPH), and residual range petroleum hydrocarbons (RRPH). Concentrations of BTEX, DRPH, and RRPH were above action levels.

A sludge pile/buried drum area was identified during the RI on the west side of the landfill, approximately 50 feet north of the gravel road. The sludge pile/buried drum area covered approximately 200 square feet. Sampling and analyses during the RI indicated that contaminants were migrating from this area towards the Arctic Ocean. The sludge pile/buried drum area was excavated as part of interim remedial actions (IRA) in May 1995. Excavated materials are temporarily stored in an onsite containment cell. Sampling and analyses conducted on soils from the sludge pile/buried drum area and the containment cell are discussed in the Final Interim Remedial Action Report for the Cape Lisburne Radar Station (U.S. Air Force 1995).

- **White Alice Site (SS03)** - This site is a communications site that was deactivated in 1979. It is located in the Upper Camp area on the southwest corner of the installation. The equipment and furniture were removed from the site in 1980, but the structures remain. The structures include an approximately 155 feet by 60 feet radio relay building and two large White Alice "billboards" that look like outdoor movie screens. This site once had transformers that contained polychlorinated biphenyl (PCB)-contaminated oils. It was suspected that dielectric fluids containing PCBs were discharged to the surrounding surface soils in small quantities during maintenance of the facility equipment.
- **Spill/Leak #3 (ST07)** - This site is located in the vicinity of the POL tanks, adjacent to the Arctic Ocean and the east end of the airstrip. The site consists of a bermed area around two large diesel tanks and the man-made hillside and drainage channel to the north. The capacity of each of the diesel tanks is approximately 490,000 gallons. In August 1992, site personnel informed the Alaska Department of Environmental Conservation (ADEC) that fuel had been



observed seeping from the north hillside, adjacent to the POL tanks. Test pits were dug and approximately 25 gallons of fuel were collected. Leak tests were conducted, and site personnel determined that the tanks were not leaking (ADEC 1992). Visual observation made during the RI at this location showed a few gallons of diesel product in an approximately 2-foot by 5-foot polyethylene plastic-lined catchment area located at the base of the hillside north of the POL tanks. Oil absorbent booms have been laid across the drainage channel to collect diesel reaching the surface water body. An interim remedial action was conducted at the site during September 1994. An interception trench and collection and treatment system were installed to reduce the migration of diesel range petroleum from the hillside below the POL tanks to the drainage channel at the toe of the hill. Interim remedial action activities conducted at the site are presented in the Final Interim Remedial Action Report for the Cape Lisburne Radar Station (U.S. Air Force 1995).

- **Upper Camp Transformer Building (SS08)** - This site is located east of the radome in the Upper Camp. The site consists of an approximately 35 feet by 45 feet building placed on gravel pad and bedrock. The building has a gravel floor with a concrete pad in the center. The transformers have been removed from the building; however, staining is apparent on the concrete pad and on the adjacent soil.
- **Lower Camp Transformer Buildings (SS09)** - This site is composed of two transformer buildings placed on a gravel pad located approximately 100 feet northwest of the main composite building. Both buildings have a gravel floor with a concrete pad in the center. Staining is apparent on the concrete pads and on the adjacent soils in both buildings. Station personnel indicate that the buildings had contained several PCB-bearing electrical transformers. The transformers have been removed from the building, but some support structures still remain.

Table 1-1 (page 1-5) contains a summary of the environmental media sampled at each of these sites. The analytical data obtained from these samples form the basis of the human health and ecological risk assessments presented in this document. Figures of the five sites are presented in Section 2.0.

#### **1.4 APPROACH TO HUMAN HEALTH RISK ASSESSMENT**

The Cape Lisburne radar installation presents a unique challenge in the development of a human health risk assessment. Many of the conventional assumptions applied in risk assessments do not apply to the North Slope of Alaska. Cape Lisburne is remote and sparsely populated. Native residents of the North Slope, largely Inupiat, follow a lifestyle that includes a significant subsistence component; much of their food consists of mammals (whales, moose, seals, and caribou), aquatic life (arctic char), and birds (ptarmigan and ducks) that are abundant in this area of the arctic. The climate is generally harsh, and the soil and surface water are frozen for approximately nine months of the year.

The general approach to the human health risk assessment was to quantify the excess lifetime cancer risk or the noncancer hazard for the site contaminants detected at each of the five sites at the installation. The maximum concentration of each chemical detected was used instead of an arithmetic mean or 95th percentile upper confidence limit (UCL) because contamination was detected infrequently and found to be generally of low concentration. Incorporating nondetects into the calculation of an average or UCL when the frequency of positive detects is low tends to yield low and unreliable estimates of contamination. Use of the maximum concentration yields a conservative estimate of risk or hazard.

To the extent possible, site-specific information was incorporated into the development of the exposure assumptions. The harsh climate naturally serves to limit exposure to contaminated soil, sediment, and surface water.

Residential exposure assumptions were used to reflect the upper-bound potential future risk. Several North Slope communities have requested use of inactive buildings at radar installations; therefore, an evaluation using potential future residential scenarios at the installations and sites was conducted.

Excess lifetime cancer risk and noncancer hazard were calculated for the soil/sediment ingestion and water ingestion pathways. Other pathways were eliminated from consideration as described in Section 2.2, the Human Health Risk Exposure Assessment.

## **1.5 APPROACH TO ECOLOGICAL RISK ASSESSMENT**

The objective of the ERA is to estimate potential impacts to aquatic and terrestrial plants and animals at the Cape Lisburne radar installation. The MITRE guidance (1990) suggests that ERAs should "estimate the potential for occurrence of adverse effects that are manifested as changes in the diversity, health and behavior" of ecosystems. MITRE proposes that this can be accomplished by:

- Estimating the health risk to individual species;
- Evaluating the health of the community of exposed species; and
- Determining the potential adverse effects of contamination over several life cycles of the species under study.

Because this is a screening level assessment, the scope of this ERA is limited to the first task: estimating the risk to individual species. If a potential risk to individual species is identified, further work may be recommended to evaluate the community and life cycle effects. It is important to note that the health risk to an individual species is different from the health risk to an individual within a species. The former refers to population level biology, where the individual is not considered a relevant endpoint. The latter assesses the risks to an individual. In this ecological assessment, the individual is considered only in the case of threatened or endangered species.

## **2.0 BASELINE HUMAN HEALTH RISK ASSESSMENT**

The purpose of the baseline human health risk assessment for the Cape Lisburne radar installation is to provide a basis for developing a risk management plan, including remedial action alternatives based on data from the RI/FS. The risk assessment develops numerical estimates of cancer risk and noncancer hazard for each site where sufficient information is available. Where information is not adequate to quantify noncancer hazard or cancer risk for a given COC, a qualitative discussion of the toxicity of that COC is provided in the Toxicity Profiles (Appendix B).

This baseline human health risk assessment addresses issues unique to this location as described in the introduction. It follows the conventional approach in that it is comprised of six sections:

- Identification of COCs - in which the chemicals detected in environmental samples are compared to risk-based screening levels (RBSLs) and concentrations considered to be applicable or relevant and appropriate requirements (ARARs);
- Exposure assessment - in which the frequency, duration, and magnitude of potential exposures to the COCs are estimated;
- Toxicity assessment - in which the toxicology of the COCs is assessed;
- Risk characterization - in which the potential for adverse health effects in humans as a result of exposure to the COCs is quantified (as appropriate) or qualitatively discussed;
- Uncertainty assessment - in which the general sources of uncertainty in the risk assessment process and the site-specific sources of uncertainty are discussed; and
- Risk assessment summary and conclusions - in which the human health risks at each of the sites are summarized and conclusions based on these risks are presented.

### **2.1 IDENTIFICATION OF CHEMICALS OF CONCERN**

COCs for human health were selected for each site at the Cape Lisburne installation based on comparison of chemical concentrations to RBSLs, naturally-occurring background concentrations, ARARs, and safe levels of essential human nutrients (e.g., calcium, magnesium, sodium, and potassium).

This section discusses the RI sampling strategy and an evaluation of data prior to screening (Section 2.1.1), describes and presents equations for calculating RBSLs (Section 2.1.2), identifies

chemicals that are essential human nutrients (Section 2.1.3), describes the collection of background samples (Section 2.1.4), and discusses the selection of COCs (Section 2.1.5).

### **2.1.1 Sampling Strategy and Evaluation of Analytical Data**

The RI sampling strategy at the Cape Lisburne sites was to characterize the nature and extent of potential contamination at each site. Suspected source areas were sampled to determine the concentrations of contaminants, if any, at the areas likely to have the highest concentrations. Migration pathways from the source areas were sampled to determine the extent, if any, that the contaminants had migrated from the sites. If no discernable pathways were evident, an attempt was made to sample around the source areas to determine the extent of site contaminants. Quick turn-around analyses were conducted on samples from the first sampling event, and a second round of sampling was conducted at those sites where further characterization of the nature and extent of contamination was needed.

Sample types included surface and subsurface soil/sediment samples and surface water samples. In almost all cases, samples were discrete grab samples from one sample location. Surface soil and sediment samples were collected in gravel and tundra areas at or near the ground surface (from ground surface to approximately six inches in depth). Subsurface soil samples were mainly collected in gravel pad areas where unsaturated conditions allowed vertical migration of contaminants. Sediment samples were collected below shallow ponds or streams, or in areas that visually appeared to have been previously covered with water. Surface water samples were collected from ponds, streams, springs, or seep areas. Surface water samples underwent both total and dissolved metal analyses; however, the total metal analytical results were used in the risk assessment. A summary of the 1993 RI sampling and analyses conducted at the installation is presented in Appendix G (includes further characterization sampling and analyses conducted in 1994 and 1995).

An IRA was conducted at the Landfill and Waste Accumulation Area in September 1994 and May 1995. The sludge pile/buried drum area was excavated and placed in an onsite containment cell. Because these wastes are currently contained, analytical results from sampling at the sludge pile/buried drum area are not used in the human health and ecological risk assessment.

Before screening for COCs, the results of the RI sampling program were sorted by medium (i.e., soil, sediment, and surface water) and reviewed for quality. The review included an evaluation of the analytical methods used, sample quantitation limits, and qualified data, and a comparison to background levels and laboratory and field blanks. Analytical data were reviewed for completeness, comparability, representativeness, precision, and accuracy. In addition, data validation qualifiers were considered in assessing the quality of the data. The review and validation of analytical data determined that a minimal amount of data was not usable. These data were qualified with an "R" and were not used in the risk assessment.

As outlined in the Risk Assessment Guidance for Superfund (EPA 1989a), site data were compared to available blank (laboratory, field, and trip) data. The data from blanks are presented in Appendix G. In accordance with EPA (1989a), if the detected concentration in a sample was less than 10 times the concentration from blanks for common laboratory contaminants (e.g.,

acetone, 2-butanone, methylene chloride, toluene, and the phthalate esters) the chemical was not selected for evaluation in the risk assessment. For those organic or inorganic chemicals that are not considered by EPA to be common laboratory contaminants (all other compounds), if the detected concentration was less than five times the maximum concentration detected in the blanks, the chemical was not selected for evaluation in the risk assessment.

## 2.1.2 Risk-Based Screening Levels

An RBSL is a chemical concentration in a particular medium that yields a given cancer risk or hazard quotient (HQ) (e.g.,  $10^{-7}$  cancer risk; 0.1 HQ) under a given set of conditions. For Cape Lisburne, the RBSLs were calculated for soil based on EPA default reasonable maximum exposure parameters (EPA 1991a). In developing the RBSLs, the most recent toxicity factors available from the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST) were used. IRIS and HEAST are databases of toxicity information for human health risk assessment maintained by the Environmental Criterion Assessment Office (ECAO) of the EPA. The information presented on IRIS represents the consensus of EPA scientists regarding the toxicity of chemicals released to the environment. Toxicity factors that EPA has withdrawn from IRIS and HEAST, or available from other sources were not used in this risk assessment.

**2.1.2.1 Formulae for Calculating RBSLs.** The RBSL concentrations were derived using equations in EPA Region 10 guidance (EPA 1991a). These equations are also presented in a slightly different form in the Risk Assessment Guidance for Superfund Volume I, Part B (EPA 1991b). Exposure assessment and risk characterization algorithms for human health risk assessments use site-specific contaminant concentration data, factors describing exposure, and toxicity dose-response values [e.g., reference doses (RfDs) or carcinogen slope factors]. These risk assessment algorithms were solved for the concentration term to derive the RBSL for soil and ground or surface water. The algorithms are summarized as follows:

$$\text{Risk} = C \times \left( \frac{CR \times EFD}{BW \times AT} \right) \times SF \quad \text{or} \quad \text{HQ} = C \times \left( \frac{CR \times EFD}{BW \times AT} \right) / \text{RfD} \quad \text{EQUATION 1, 2}$$

Risk = Target Cancer Risk

C = Concentration

CR = Contact Rate

EFD = Exposure Frequency and Duration

BW = Body Weight

AT = Averaging Time

SF = Slope Factor

HQ = Target Hazard Quotient

RfD = Reference Dose

RBSLs are calculated based on a specific target cancer risk or HQ. EPA (1991a) recommends that a  $1 \times 10^{-7}$  target cancer risk and a target noncancer HQ of 0.1 be used for soil and a  $1 \times 10^{-6}$  risk and 0.1 HQ be used for surface water. The lower target cancer risk is used for screening soil because additional pathways, such as dermal contact and inhalation, are not accounted for by the calculations (EPA 1991a).

Equations (1) and (2) shown above are rearranged to solve for the concentration term (i.e., the RBSL):

$$C = \text{Risk} / \left( \left( \frac{CR \times EFD}{BW \times AT} \right) \times SF \right) \quad \text{or} \quad C = HQ / \left( \left( \frac{CR \times EFD}{BW \times AT} \right) / RfD \right) \quad \text{EQUATION 3, 4}$$

**Surface Water Ingestion Equations.** Using standard default exposure factors (EPA 1989b) for water ingestion, the equation for cancer risk from drinking water ingestion becomes:

$$\text{Risk} = C (\mu\text{g/L}) \times 0.001 \text{ mg}/\mu\text{g} \times \left( \frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 70 \text{ year} \times 365 \text{ day/year}} \right) \times SF_o \quad \text{EQUATION 5}$$

Equation 5 can be rearranged to solve for a RBSL with, for example, a target cancer risk of  $10^{-6}$ :

$$C (\mu\text{g/L}) = 10^{-6} \times 1,000 \mu\text{g}/\text{mg} / \left[ \left( \frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 70 \text{ year} \times 365 \text{ day/year}} \right) \times SF_o \right] \quad \text{EQUATION 6}$$

For non-carcinogens, the equation for the HQ for drinking water ingestion is:

$$HQ = C (\mu\text{g/L}) \times 0.001 \text{ mg}/\mu\text{g} \times \left( \frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 30 \text{ year} \times 365 \text{ day/year}} \right) / RfD_o \quad \text{EQUATION 7}$$

Equation 7 can be rearranged to provide an equation for the concentration that represents an HQ of 1 from ingestion:

$$C (\mu\text{g/L}) = 1 \times 1,000 \mu\text{g}/\text{mg} / \left[ \left( \frac{2 \text{ L/day} \times 350 \text{ day/year} \times 30 \text{ year}}{70 \text{ kg} \times 30 \text{ year} \times 365 \text{ day/year}} \right) / RfD_o \right] \quad \text{EQUATION 8}$$

**Soil or Sediment Ingestion Equations.** The equation for calculating carcinogenic risk from soil or sediment ingestion, combining child and adult exposure, is as follows:

$$\text{Risk} = C (\text{mg}/\text{kg}) \times 0.000001 \text{ kg}/\text{mg} \times \quad \text{EQUATION 9}$$

$$\left[ \left( \frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left( \frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) \right] / 70 \text{ year} \times SF_o$$

Equation 9 can be rearranged to solve for the concentration that represents a target cancer risk of  $10^{-7}$ :

$$C (\text{mg}/\text{kg}) = 10^{-7} \times 1,000,000 \text{ kg}/\text{mg} / \quad \text{EQUATION 10}$$

$$\left[ \left( \frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left( \frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) \right] / 70 \text{ year} \times SF_o$$

For non-carcinogens in soil, Equation 11 is used to calculate the HQ:

$$HQ = C \text{ (mg/kg)} \times 0.000001 \text{ kg/mg} \times$$

EQUATION 11

$$\left[ \left( \frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left( \frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) / 30 \text{ year} \right] / RfD_o$$

Equation 12 can be rearranged to solve for the concentration that represents an HQ of 0.1:

$$C \text{ (mg/kg)} = 0.1 \times 1,000,000 \text{ mg/kg} /$$

EQUATION 12

$$\left[ \left( \left( \left( \frac{200_c \text{ mg/day} \times 350_c \text{ day/year} \times 6 \text{ year}}{15_c \text{ kg} \times 365 \text{ day/year}} \right) + \left( \frac{100_a \text{ mg/day} \times 350_a \text{ day/year} \times 24 \text{ year}}{70_a \text{ kg} \times 365 \text{ day/year}} \right) \right) / 30 \text{ year} \right) / RfD_o \right]$$

### 2.1.3 Screening of Chemicals by Comparing Maximum Detected Concentrations of Essential Human Nutrients

Based on EPA's guidance (1991a), calcium, magnesium, potassium, iron, and sodium are considered to be essential human nutrients and were eliminated from the human health risk assessment at the screening stage. These chemicals are often detected but are not toxic to humans except at extremely high doses. No quantitative toxicity information is available for these elements from EPA sources; therefore, these metals are not selected as COCs for this risk assessment.

### 2.1.4 Concentrations of Organic and Inorganic Constituents in Background Samples

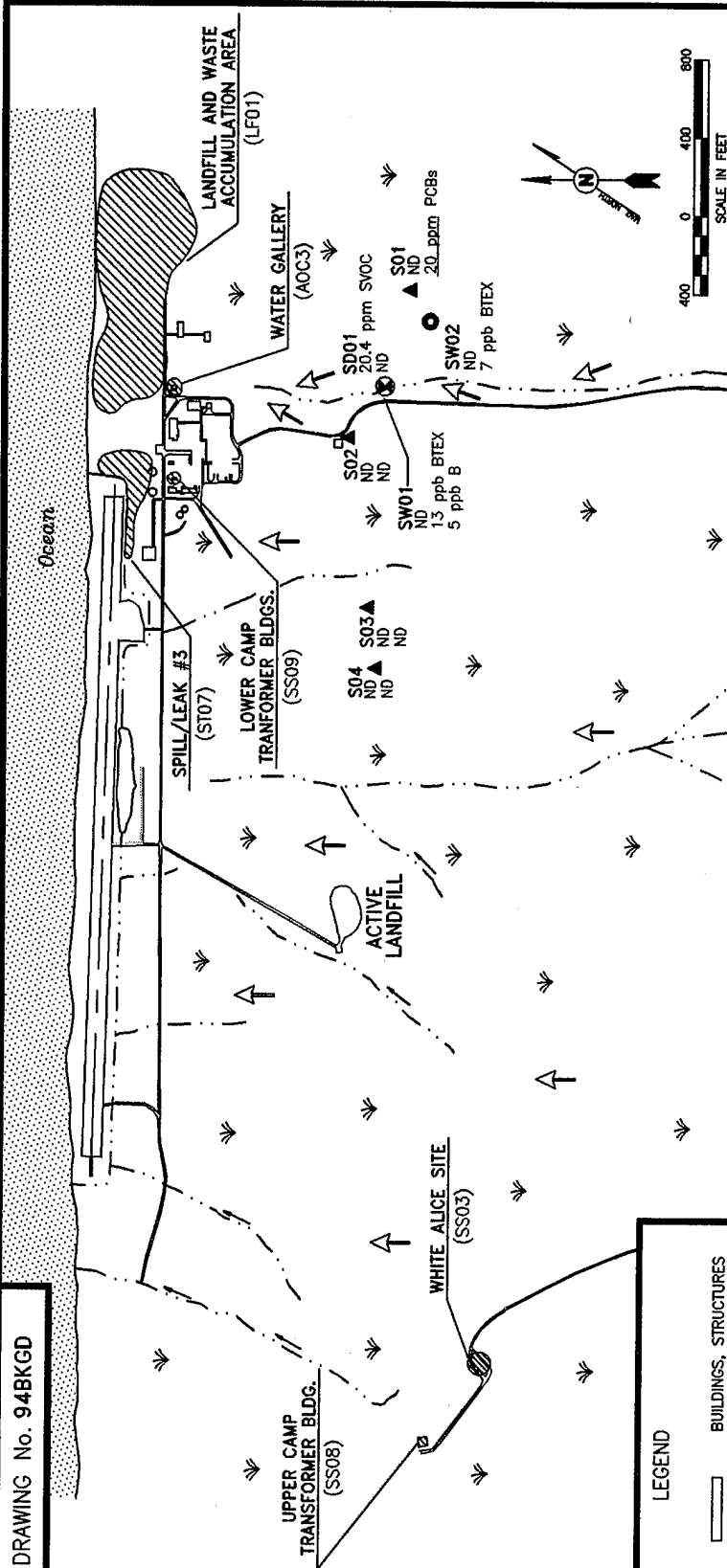
Four soil, one sediment, and two surface water samples were collected upgradient (relative to surface drainage) of the radar installation to determine the background concentrations of naturally occurring organic and inorganic constituents in soil, sediment, and surface water (Figure 2-1). Soil and sediment background samples were collected at a depth of zero to six inches. Although some compounds were detected in some of the soil and sediment background samples in the DRPH and PCB analyses, the organic concentration in background samples is assumed to be non-detect. This conservative approach was used because it is not possible to determine to what degree, if any, the DRPH detected in site samples were naturally occurring compounds. The detection of PCBs in one background sample may be due to laboratory or field cross-contamination.

To obtain a representative range of background inorganic (metal) concentrations in soil, sediments, and surface waters at Cape Lisburne, seven samples (the same five soil/sediment and two water samples mentioned above) were analyzed for metals. Total metal concentrations in surface water were used in this risk assessment.

THIS PAGE INTENTIONALLY LEFT BLANK



DRAWING No. 94BKGD



# LEGEND

- BUILDINGS, STRUCTURES
- ROADS
- 94 SOIL SAMPLE
- TUNDRA
- SURFACE WATER SAMPLE
- SEDIMENT/WATER SAMPLE
- SURFACE WATER
- RIVER, STREAM, OR CREEK
- SURFACE DRAINAGE
- CT&E DATA
- F&B DATA
- RI SITES AND THE AREA OF CONCERN

CONCENTRATIONS ARE ABOVE GUIDANCE CLEANUP LEVELS

ND NO CONTAMINATION DETECTED

SVOC TOTAL SEMI-VOATILE ORGANIC COMPOUNDS

B BENZENE

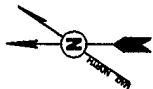
PCBs POLYCHLORINATED BIPHENYLS

## CAPE LISBURNE RADAR INSTALLATION

### USAF 611th CES

FIGURE NO. 2-1

BACKGROUND (BKGD)  
SAMPLE LOCATIONS  
AND ORGANIC  
ANALYTICAL RESULTS



THIS PAGE INTENTIONALLY LEFT BLANK

### 2.1.5 Selection of Chemicals of Concern

**Soil and Sediment.** The maximum concentration of the chemicals detected in soil or sediment samples at the Cape Lisburne installation and not considered to be essential human nutrients were compared, on a site-by-site basis, to the corresponding background concentrations, RBSLs, and where available, federal or state ARARs. Chemicals detected without an RBSL or ARAR were retained as COCs if concentrations exceeded background levels. A chemical with an RBSL or ARAR was selected as a COC for soil and sediment if the maximum concentration at which the chemical was detected exceeded the corresponding background concentration and the RBSL (based either on cancer risk or noncancer hazard) or ARAR (Table 2-1). Thus, for example, the maximum concentration of Aroclor 1260 (6,290 mg/kg) at the White Alice Site (SS03) exceeds the background range, the RBSLs based on cancer risk and noncancer hazard, and the ARAR for PCBs promulgated in the Toxic Substances Control Act (TSCA). Therefore, Aroclor 1260 would be selected as a COC for the soils at the White Alice Site.

The soil/sediment COCs at each site were compared to background concentrations, RBSLs, and ARARs in Table 2-1. The chemicals retained as COCs exceed background concentrations, the RBSL, or an ARAR. A chemical was not retained if the level detected was less than the corresponding RBSL and ARAR, even though background levels were exceeded. The COCs selected that do not have an RBSL or an ARAR are discussed below. The COCs selected at each site that exceed an RBSL, ARAR, or both, are discussed in Sections 2.1.5.1 to 2.1.5.5.

**Surface Water.** The maximum concentrations of the chemicals detected in surface water samples at the Cape Lisburne installation were compared, on a site-by-site basis, to the corresponding background concentrations, RBSLs, and where available, federal or state ARARs. Chemicals detected without an RBSL or ARAR were retained as COCs if concentrations exceeded background levels. A chemical with an RBSL or ARAR was selected as a COC for surface water if the maximum concentration at which the chemical was detected exceeded the corresponding background concentration, and the RBSL (based either on cancer risk or noncancer hazard) or ARAR (Table 2-1). Thus, for example, the maximum concentration of carbon tetrachloride (89 µg/L) at the Landfill and Waste Accumulation Area (LF01) exceeds the RBSL based on noncancer hazard (2.56 µg/L), the RBSL based on cancer risk (0.65 µg/L), and the ARAR (5 µg/L), which is an MCL promulgated under the federal Safe Drinking Water Act. Therefore, carbon tetrachloride was selected as a COC for the surface water at the Landfill and Waste Accumulation Area (LF01).

The surface water COCs at each site were compared to background concentrations, RBSLs, and ARARs in Table 2-1. The chemicals retained as COCs exceed background concentrations, the RBSL, or an ARAR. A chemical was not retained if the level detected was less than the corresponding RBSL and ARAR, even though background levels were exceeded. The COCs selected that exceed background levels, but do not have an RBSL or ARAR are discussed below. The COCs at each site that exceed an RBSL, ARAR, or both, are discussed in Sections 2.1.5.1 to 2.1.5.5.

**TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT CAPE LISBURNE**

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL <sup>a</sup>		ARAR <sup>b</sup>	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Landfill and Waste Accumulation Area (LF01)	Soil or Sediments	DRPH	18,600	mg/kg	<60-<150	--	--	500 <sup>c</sup>	Yes
		GRPH	50.9	mg/kg	<2J-<6J	--	--	100 <sup>c</sup>	No
		RPH	43,100	mg/kg	<120-<300	--	--	2,000 <sup>c</sup>	Yes
		Aroclor 1254	0.235	mg/kg	<0.02-<0.3	0.00831 <sup>d</sup>	0.54	10 <sup>f</sup>	Yes
		Aroclor 1260	999	mg/kg	<0.02-20JN	0.00831 <sup>d</sup>	0.54 <sup>e</sup>	10 <sup>f</sup>	Yes
		Benzene	0.256	mg/kg	<0.02-<0.160	2.21	--	0.5 <sup>c</sup>	No
		n-Butylbenzene	0.636	mg/kg	<0.025-<0.160	--	--	--	Yes*
		sec-Butylbenzene	0.168	mg/kg	<0.025-<0.160	--	--	--	Yes*
		Carbon tetrachloride	17.3	mg/kg	<0.02J-<0.160	0.492	18.9	--	Yes
		Chloroform	0.796	mg/kg	<0.025-<0.160	10.5	270	--	No
		cis-1,2-Dichloroethene	0.048	mg/kg	<0.025-<0.160	--	270	--	No
		Ethylbenzene	2.19	mg/kg	<0.02-<0.160	--	2,700	--	No
		Isopropylbenzene	0.242	mg/kg	<0.025-<0.160	--	--	--	Yes*
		p-Isopropyltoluene	0.178	mg/kg	<0.025-<0.160	--	--	--	Yes*
		Methylene chloride	0.066	mg/kg	<0.02J-<0.160	8.53	1,620	--	No
		2-Methylnaphthalene	8.59	mg/kg	<0.25-<4.23	--	--	--	Yes*
		4-Methylphenol	2.16	mg/kg	<0.25-<4.23	--	135	--	No
		Naphthalene	4.26J	mg/kg	<0.25-<4.23	--	1,100	--	No
		Phenol	0.920	mg/kg	<0.25-<4.23	--	16,200	--	No
		n-Propylbenzene	0.758	mg/kg	<0.025-<0.160	--	--	--	Yes*
		Toluene	8.45	mg/kg	<0.02-<0.160	--	5,400	--	No
		Trichloroethene	15.3	mg/kg	<0.025-<0.160	5.8	--	--	Yes
		1,2,4-Trimethylbenzene	6.89	mg/kg	<0.025-<0.160	--	--	--	Yes*
		1,3,5-Trimethylbenzene	2.04	mg/kg	<0.025-<0.160	--	--	--	Yes*

**TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)**

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL <sup>a</sup>		ARAR <sup>b</sup>	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Landfill and Waste Accumulation Area (LFO1) (Continued)	Soil or Sediments (Continued)	Xylenes	9.83	mg/kg	<0.04-<0.320	--	54,000	--	No
		Aluminum	6,000	mg/kg	4,700-17,000	--	--	--	No
		Barium	630	mg/kg	590-2,000	--	1,890	--	No
		Calcium	41,000	mg/kg	2,700-240,000	--	--	--	No
		Chromium	9.8	mg/kg	9.3-33	--	135	--	No
		Cobalt	5.6J	mg/kg	<6.3-17	--	--	--	No
		Copper	13	mg/kg	12-71	--	999	--	No
		Iron	18,000	mg/kg	5,400-39,000	--	--	--	No
		Magnesium	11,000	mg/kg	1,000-34,000	--	--	--	No
		Manganese	750J	mg/kg	15J-1,000J	--	3,780	--	No
		Nickel	17	mg/kg	13-80	--	540	--	No
		Potassium	1,370	mg/kg	<540-2,600	--	--	--	No
		Sodium	290	mg/kg	58-120	--	--	--	No
		Vanadium	18	mg/kg	19-58	--	189	--	No
		Zinc	73	mg/kg	40J-250	--	8,100	--	No
	Surface Water <sup>d</sup>	Benzene	5J	µg/L	<1	0.617	--	5 <sup>g</sup>	Yes
		Carbon tetrachloride	89J	µg/L	<0.02J-<1	0.026	2.56	5 <sup>g</sup>	Yes
		Chloroform	16J	µg/L	<1	1.4	36.5	100 <sup>i</sup>	Yes
		cis-1,2-Dichloroethene	1.5	µg/L	<1	--	36.5	70	No
		Ethylbenzene	2	µg/L	<1	--	158	700 <sup>h</sup>	No
		Tetrachloroethene	4J	µg/L	<0.02J-<1	0.143	36.5	5 <sup>h</sup>	Yes
		Toluene	9.6J	µg/L	<1	--	96.5	1,000 <sup>h</sup>	No
		Trichloroethene	62J	µg/L	<0.02J-<1	0.25	--	5 <sup>g</sup>	Yes
		Xylene	5J	µg/L	<2	--	7,300	10,000 <sup>h</sup>	No

**TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)**

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL <sup>a</sup>		ARAR <sup>b</sup>	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Landfill and Waste Accumulation Area (LF01) (Continued)	Surface Water (Continued)	Aluminum	160	µg/L	<100	--	--	--	Yes*
		Barium	860	µg/L	79-92	--	256	2,000 <sup>i</sup>	Yes
		Calcium	150,000	µg/L	28,000-41,000	--	--	--	No
		Iron	4,900	µg/L	<100-190	--	--	--	No
		Magnesium	30,000	µg/L	4,500-9,000	--	--	--	No
		Manganese	1,800	µg/L	<50	--	18.3	--	Yes
		Sodium	38,000	µg/L	4,000-5,600	--	--	--	No
		DRPH	380J	mg/kg	<60-<150	--	--	500 <sup>c</sup>	No
White Alice Site (SS03)	Soil or Sediments	Aroclor 1260	6,290	mg/kg	<0.1-20JN	0.00831 <sup>d</sup>	0.540 <sup>e</sup>	10 <sup>f</sup>	Yes
NO SURFACE WATER BODIES LOCATED AT WHITE ALICE SITE (SS03)									
Spill/Leak #3 (ST07)	Soil or Sediment	DRPH	63,000	mg/kg	<60-<150	--	--	500 <sup>c</sup>	Yes
		GRPH	150	mg/kg	<2J-<6J	--	--	100 <sup>c</sup>	Yes
		RRPH	1,200	mg/kg	<120-<300	--	--	2,000 <sup>c</sup>	No
		Benzene	1.4J	mg/kg	<0.02-<0.160	2.21	--	0.5 <sup>c</sup>	Yes
		sec-Butylbenzene	0.37J	mg/kg	<0.025-<0.160	--	--	--	Yes*
		1,4-Dichlorobenzene	0.095	mg/kg	<0.025-<0.160	--	0.267	--	No
		Ethylbenzene	7	mg/kg	<0.02-<0.160	--	2,700	--	No
		Isopropylbenzene	0.21J	mg/kg	<0.025-<0.160	--	--	--	Yes*
		p-Isopropyltoluene	0.762J	mg/kg	<0.025-<0.160	--	--	--	Yes*
		2-Methylnaphthalene	5.12J	mg/kg	<0.250-<4.23	--	--	--	Yes*
		4-Methylphenol	7.32J	mg/kg	<0.250-<4.23	--	135	--	No
		Naphthalene	4.18J	mg/kg	<0.025-<4.23	--	1,100	--	No
		n-Propylbenzene	0.366J	mg/kg	<0.025-<0.160	--	--	--	Yes*
		Toluene	0.949	mg/kg	<0.02-<0.160	--	5,400	--	No

**TABLE 2-1. IDENTIFICATION OF CHEMICALS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)**

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL <sup>a</sup>		ARAR <sup>b</sup>	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Spill/Leak #3 (ST07) (Continued)	Soil or Sediment (Continued)	Trichloroethene	0.426	mg/kg	<0.02J-<0.160	5.8	--	--	No
		1,2,4-Trimethylbenzene	2.81	mg/kg	<0.025-<0.160	--	--	--	Yes*
		1,3,5-Trimethylbenzene	1.76	mg/kg	<0.025-<0.160	--	--	--	Yes*
		Xylene	10J	mg/kg	<0.04-<0.320	--	54,000	--	No
	Surface Water	Naphthalene	1.7	µg/L	<1	--	146	--	No
Upper Camp Transformer Building (SS08)	Soil or Sediment	1,2,4-Trimethylbenzene	1.2	µg/L	<1	--	--	--	Yes*
		DRPH	51,000J	mg/kg	<60-<150	--	--	500 <sup>c</sup>	Yes
		RRPH	29,000	mg/kg	<120-<300	--	--	2,000 <sup>c</sup>	Yes
		Aroclor 1254	29.1	mg/kg	<0.02-<0.3	0.00831 <sup>d</sup>	0.540	10 <sup>f</sup>	Yes
		Aroclor 1260	300JN	mg/kg	<0.1-20JN	0.00831 <sup>d</sup>	0.540 <sup>e</sup>	10 <sup>f</sup>	Yes
NO SURFACE WATER BODIES LOCATED AT UPPER CAMP TRANSFORMER BUILDING (SS08) SITE									
Lower Camp Transformer Buildings (SS09)	Soil or Sediment	DRPH	30,000J	mg/kg	<60-<150	--	--	500 <sup>c</sup>	Yes
		RRPH	13,000	mg/kg	<120-<300	--	--	2,000 <sup>c</sup>	Yes
		Aroclor 1254	1,720	mg/kg	<0.02-<0.3	0.00831 <sup>d</sup>	0.540	10 <sup>f</sup>	Yes
		Aroclor 1260	5,600	mg/kg	<0.02-20JN	0.00831 <sup>d</sup>	0.540 <sup>e</sup>	10 <sup>f</sup>	Yes
NO SURFACE WATER BODIES LOCATED AT LOWER CAMP TRANSFORMER BUILDINGS (SS09) SITE									

\* Chemicals without an RBSL or ARAR are considered chemicals of potential concern and are discussed in Section 2.1.5.

< Indicates not detected at reported quantitation limit.

<sup>a</sup> Risk-Based Screening Level.

<sup>b</sup> Applicable or Relevant and Appropriate Requirement.

<sup>c</sup> Target cleanup levels for DRPH, GRPH, and RRPH in soil are based on ADEC Non-UST guidance and do not necessarily correspond to final site specific cleanup goals (ADEC 1991).

<sup>d</sup> Cancer RBSL for Aroclor 1254 and 1260 is based on the cancer slope factor for PCBs (IRIS 1995).

<sup>e</sup> Noncancer RBSL for Aroclor 1260 based on oral RfD for Aroclor 1254.

<sup>f</sup> TSCA cleanup level.

<sup>g</sup> MCL, 52 FR 25690 (08 Jul 87).

<sup>h</sup> MCL, 56 FR 3526 (30 Jan 91).

<sup>i</sup> MCL, 56 FR 30266 (01 Jul 91).

<sup>j</sup> The concentrations reported for metals in surface water are results from total metal analyses.

<sup>k</sup> Result is an estimate.

<sup>n</sup> Presumptive evidence of presence of material (tentative identification).

**Risk Characterization of Chemicals without RBSLs and ARARs.** Several chemicals detected above background levels could not be thoroughly screened because an RBSL could not be calculated and no ARAR was available (Table 2-1). A list of these chemicals is presented in Table 2-2. The cancer risk and noncancer hazard for these chemicals cannot, therefore, be quantified. This section is a qualitative discussion of the potential for these chemicals to cause toxicity among the receptor groups identified at the Cape Lisburne installation. The essential human nutrients were discussed in Section 2.1.3 and will not be discussed further here. Essential nutrients are not considered COCs in this risk assessment.

The American Petroleum Institute (API) recently published an evaluation of the environmental fate, transport, and toxicity of twelve organic chemicals found frequently in petroleum products. The twelve were selected from a large list of "candidates" based on:

- abundance in crude and refined petroleum products, including residual and used oils;
- chemical/physical properties that represent a range of mobilities in soil and solubilities in aqueous environments; and
- toxicity in mammals and aquatic organisms (API 1994).

Two of the chemicals detected at Cape Lisburne installation, 1,2,4-trimethylbenzene and naphthalene, were selected from the list of twelve chemicals (API 1994) and are used in this risk assessment as surrogates for the chemicals without RBSLs and ARARs. These chemicals have similar chemical structures and, therefore, will represent the substituted benzenes and the polynuclear aromatic hydrocarbons that do not have toxicity criteria (Table 2-2).

1,2,4-Trimethylbenzene has a low order of toxicity in mammals (API 1994). No effect was observed on the kidneys of rats that received 0.5 or 2.0 g/kg orally five days per week for four weeks. Inhalation of high concentrations of 1,2,4-trimethylbenzene produces central nervous system depression in humans and rats. Lung toxicity, including bronchitis, pneumonitis, and edema, was also observed in humans. 1,2,4-Trimethylbenzene has not been observed to be carcinogenic or mutagenic in laboratory studies of rats and cultured mammalian cells. Potential exposure of receptors to 1,2,4-trimethylbenzene at the Cape Lisburne installation would probably be limited to oral ingestion of soil and at the maximum concentration measured (6.89 mg/kg soil) would be expected to be nontoxic.<sup>1</sup> For the purposes of this risk assessment, 1,2,4-trimethylbenzene is considered to be a reasonable surrogate for the substituted benzenes observed at the Cape Lisburne installation.

---

<sup>1</sup> Based on the following calculation: assume average daily soil ingestion rate of 200 mg of soil per day and 6.89 mg of 1,2,4-trimethylbenzene per kg of soil (maximum concentration measured at Cape Lisburne installation). This yields a dose of 0.00002 mg of 1,2,4-trimethylbenzene per kg body weight per day. The oral dose of 1,2,4-trimethylbenzene received by rats that showed no kidney effects was equivalent to 2,000 mg of 1,2,4-trimethylbenzene per kg body weight, which is approximately 100,000,000 times greater than the estimated dose for potential receptors at the Cape Lisburne installation.



**TABLE 2-2. CHEMICALS WITHOUT RBSLS AND ARARS OBSERVED IN THE SOIL, SEDIMENT, OR SURFACE WATER AT THE CAPE LISBURNE INSTALLATION**

SUBSTITUTED BENZENES	
1,2,4-Trimethylbenzene	
1,3,5-Trimethylbenzene	
n-Propylbenzene	
Isopropylbenzene	
n-Butylbenzene	
sec-Butylbenzene	
p-Isopropyltoluene	
ESSENTIAL HUMAN NUTRIENTS	
Calcium	
Iron	
Magnesium	
Potassium	
Sodium	
POLYCYCLIC AROMATIC HYDROCARBONS	
2-Methylnaphthalene	
OTHER	
Aluminum	

Because of the lack of toxicology information available for 2-methylnaphthalene, naphthalene will be used as a surrogate in this discussion of chemicals without RBSLs and ARARs. Naphthalene has a low order of toxicity in mammals (API 1994). The toxicology of this chemical has been well characterized in several species, including humans, rats, rabbits, and mice. The toxicity in humans is known from cases of accidental or intentional (suicide) ingestion of contaminated food or mothballs, and the most common effect is liver damage (jaundice) and destruction of red blood cells resulting in anemia. These effects occur at exposure levels that far exceed the levels to which the receptor groups at the Cape Lisburne installation could be exposed. Dose-response information is available from studies in rats, mice, and rabbits. High doses of naphthalene administered over several days to one month resulted in cataract formation and other less serious ocular effects. High doses administered over several days to three months produced mild toxic effects on the liver, lung, kidney, and immunological system. The no effect level of oral exposure in these species occurs in the range of 100 to 300 mg naphthalene per kg body weight per day (100 to 300 mg/kg/day). The oral exposure levels to 2-methylnaphthalene that may occur through soil ingestion at the Cape Lisburne installation are in the range of 0.00001 to 0.00004 mg/kg/day. Furthermore, the RBSL for naphthalene exceeds the maximum concentration of 2-methylnaphthalene. Therefore, any exposure to 2-methylnaphthalene in the soil at the Cape Lisburne installation is expected to be non-toxic.<sup>2</sup>

The metal without an RBSL and ARAR which is listed in the "Other" category of Table 2-2 is not expected to pose a significant hazard to the receptor groups identified at the Cape Lisburne installation. Aluminum was detected in a surface water sample at 160 µg/L. EPA's ECAO has issued a provisional reference dose for aluminum of 1 mg/kg/day. Based on the provisional criteria, aluminum is expected to be non-toxic<sup>3</sup> at the concentrations detected.

In conclusion, the chemicals discussed above have been marked in Table 2-1 as COCs to indicate that there is some uncertainty in screening out these chemicals. Without toxicity criteria the potential risks of these chemical cannot be quantified. However, based on the information in this section, and the concentrations measured at the sites, these chemicals are not expected to pose a health risk.

**Chemicals with RBSLs and/or ARARs.** Following are discussions of the COCs at each site that exceeded background levels and an RBSL, ARAR, or both. Table 2-3 is a summary of the COCs selected for the sites at the Cape Lisburne installation.

#### **2.1.5.1 Landfill and Waste Accumulation Area (LF01).**

**Soil or Sediment.** DRPH, RRPH, carbon tetrachloride, trichloroethene, and Aroclor 1260 were identified as COCs for the soil matrix at the Landfill and Waste Accumulation Area (LF01) (Figures 2-2 and 2-3, Table 2-1). The maximum concentrations of DRPH and RRPH exceeded their background concentrations and the ARAR concentrations for petroleum hydrocarbon

---

<sup>2</sup> Based on the following assumptions: soil ingestion rate, 200 mg/day; drinking water ingestion rate, 2 L/day; 70 kg body weight for typical receptor; maximum soil concentration of 2-methylnaphthalene, 8.59 mg/kg.

<sup>3</sup> Based on the following assumptions: drinking water ingestion rate, 2 L/day; 70 kg body weight for typical receptor; and maximum surface water concentration of 160 µg/L.

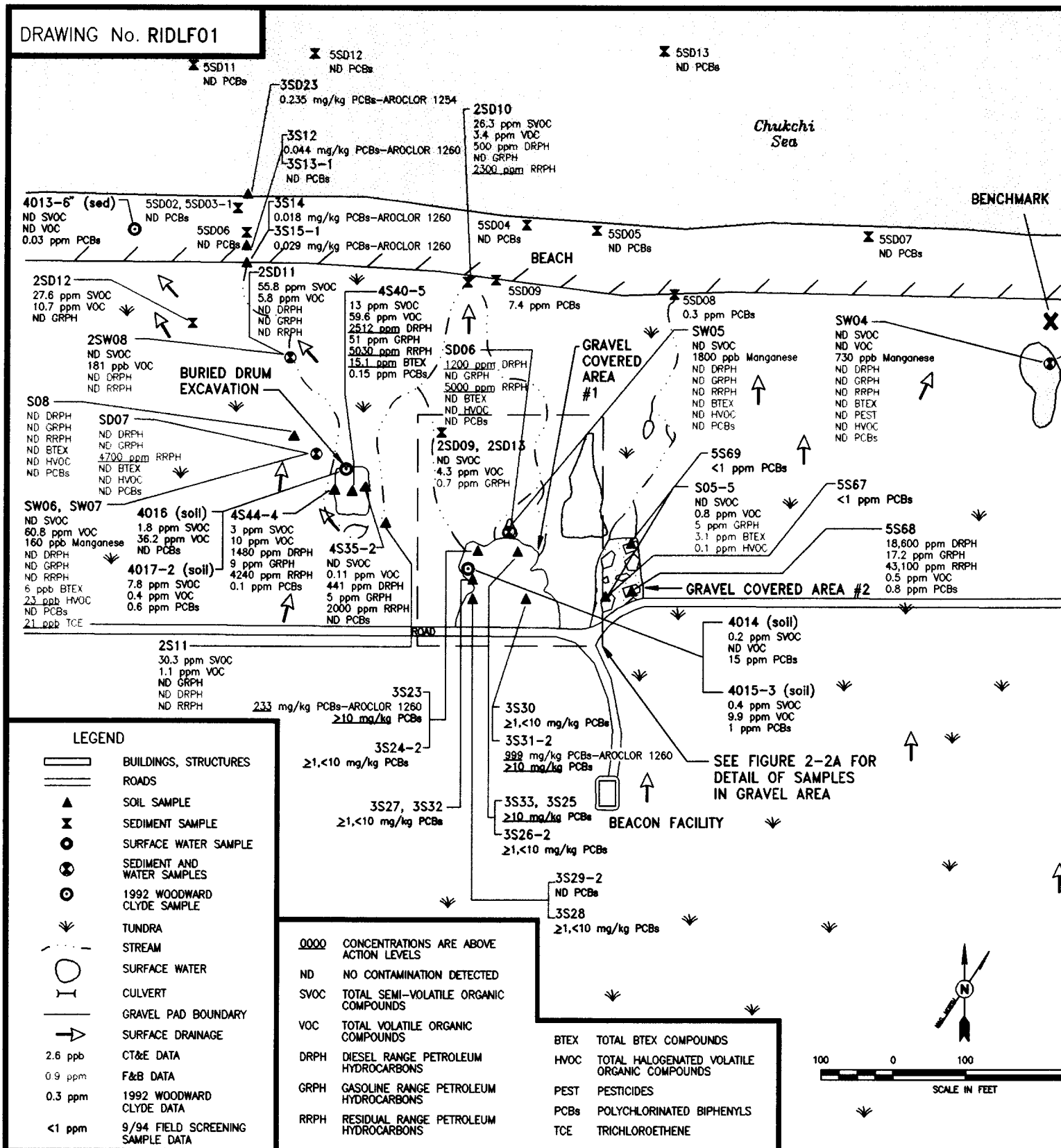
TABLE 2-3. SUMMARY OF THE CHEMICALS OF CONCERN AT CAPE LISBURN

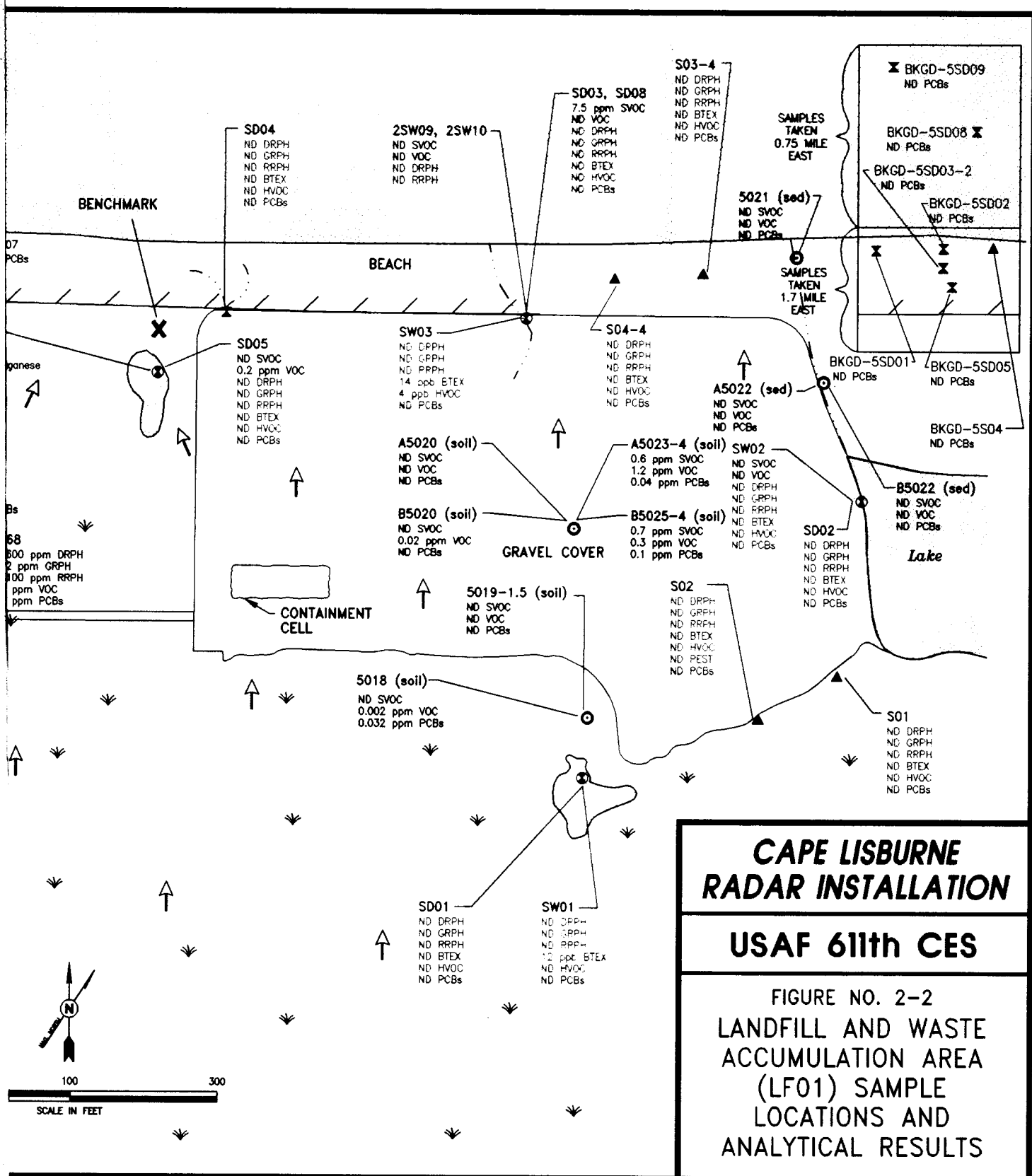
SITE	CHEMICALS OF CONCERN	
	SOIL/SEDIMENT	SURFACE WATER
Landfill and Waste Accumulation Area (LF01)	DRPH RRPH Aroclor 1260 Carbon tetrachloride Trichloroethene	Benzene Carbon tetrachloride Tetrachloroethene Trichloroethene Chloroform Barium Manganese
White Alice Site (SS03)	Aroclor 1260	NONE
Spill/Leak #3 (ST07)	DRPH GRPH Benzene	NONE
Upper Camp Transformer Building (SS08)	DRPH RRPH Aroclor 1254 Aroclor 1260	NONE
Lower Camp Transformer Buildings (SS09)	DRPH RRPH Aroclor 1254 Aroclor 1260	NONE

\* The summary of COCs on this table includes only those chemicals detected that exceed background levels and an RBSL, ARAR, or both. COCs that exceeded background levels but do not have an RBSL or ARAR are discussed in Section 2.1.5 (Page 2-9).

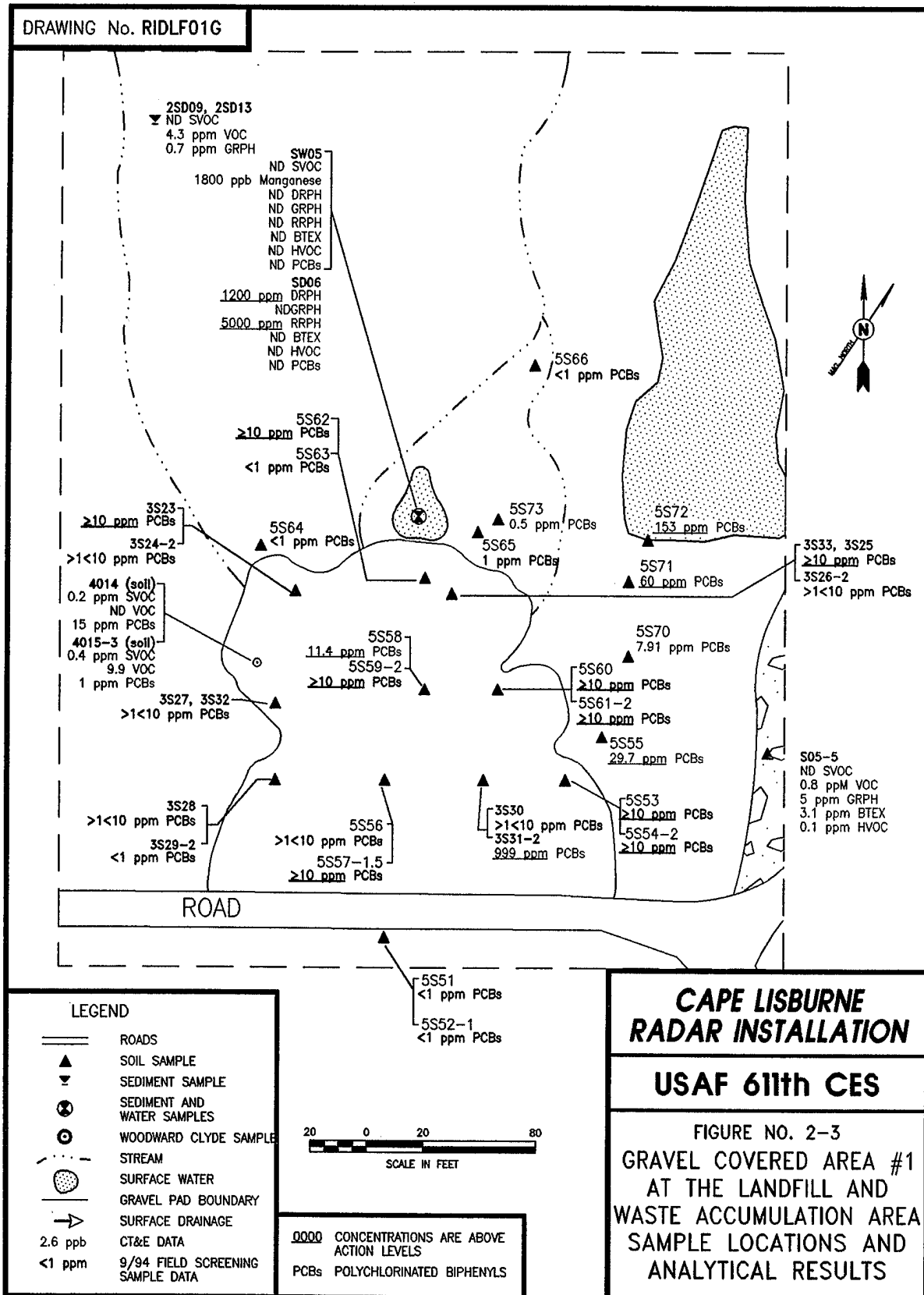
THIS PAGE INTENTIONALLY LEFT BLANK

# DRAWING No. RIDLF01





DRAWING No. RIDLF01G



THIS PAGE INTENTIONALLY LEFT BLANK



contamination of soil (ADEC 1991). Carbon tetrachloride and trichloroethene exceeded background and the RBSL based on cancer risk. Aroclor 1260 exceeded the RBSLs based on cancer risk and noncancer hazard and the ARAR for PCBs under the TSCA.

**Surface Water.** Benzene, carbon tetrachloride, chloroform, tetrachloroethene, trichloroethene, barium, and manganese were identified as COCs for the surface water at Landfill and Waste Accumulation Area (LF01) (Figures 2-2 and 2-3, Table 2-1). Benzene exceeded background and the RBSL based on cancer risk but did not exceed the ARAR, which is an MCL promulgated under the federal Safe Drinking Water Act. Carbon tetrachloride exceeded background, the RBSLs based on cancer risk and noncancer hazard, and the ARAR, which is an MCL. Tetrachloroethene exceeded background and the RBSL based on cancer risk but did not exceed the RBSL based on noncancer hazard or the ARAR, which is an MCL. Trichloroethene exceeded background, the RBSL based on cancer risk and the ARAR, which is an MCL. Chloroform exceeded background and the RBSL based on cancer risk but did not exceed the RBSL based on noncancer hazard or the ARAR which is an MCL. Barium exceeded background and the RBSL based on noncancer hazard but did not exceed the ARAR which is an MCL. Manganese exceeded background and the RBSL based on noncancer hazard.

#### **2.1.5.2 White Alice Site (SS03).**

**Soil or Sediment.** Aroclor 1260 was identified as a COC for the soil matrix at the White Alice Site (SS03) (Figure 2-4, Table 2-1). The maximum concentration of Aroclor 1260 exceeded the background concentration, the RBSLs based on cancer risk and noncancer hazard, and the ARAR for PCBs under the TSCA.

**Surface Water.** No surface water bodies were identified at the White Alice Site (SS03); therefore, no surface water COCs have been identified (Figure 2-4, Table 2-1).

#### **2.1.5.3 Spill/Leak #3 (ST07).**

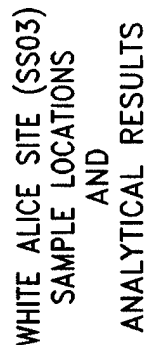
**Soil or Sediment.** DRPH, GRPH, and benzene were identified as COCs for the soil matrix at Spill/Leak #3 (ST07) (Figure 2-5, Table 2-1). DRPH and GRPH exceeded background and the ARAR concentration for petroleum hydrocarbon contamination of soil (ADEC 1991). Benzene exceeded background and the ARAR, which is a State of Alaska soil cleanup level for non-underground storage tank (UST) contaminated soils (ADEC 1991) but did not exceed the RBSL based on cancer risk.

**Surface Water.** No COCs were identified for the surface water at Spill/Leak #3 (ST07) based on a comparison of the maximum concentrations of chemicals detected to background, RBSLs, and ARARs (Figure 2-5, Table 2-1).

#### **2.1.5.4 Upper Camp Transformer Building (SS08).**

**Soil or Sediment.** DRPH, RRPH, Aroclor 1254, and Aroclor 1260 were identified as COCs for the soil matrix at the Upper Transformer Building (SS08) (Figure 2-6, Table 2-1). Each of these chemicals exceeded their respective background concentrations. DRPH and RRPH also

THIS PAGE INTENTIONALLY LEFT BLANK



THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. 94ST0717

Ocean

SW01, SW03

SD01, SD06

SD0

SD04

9.6 ppm SVOC

2.5 ppm VOC

2300 ppm DRPH

ND GRPH

ND RRPH

ND BTEX

2S15

0.2 ppm VOC

ND DRPH

ND RRPH

2.9 ppb VOC

ND DRPH

ND GRPH

ND RRPH

ND BTEX

3S22-5.5

125 mg/kg DRPH

ND BTEX

SD

ND

150

ND

RUNWAY

2SD08

0.710 ppm VOC

ND DRPH

ND RRPH

2SD07

ND VOC

ND DRPH

ND RRPH

SD05

650 ppm DRPH

20 ppm GRPH

ND RRPH

2.97 ppm BTEX

S05-3

ND DRPH

ND GRPH

ND RRPH

ND BTEX

S04-2

ND DRPH

25 ppm GRPH

ND RRPH

ND BTEX

3S21-4

1190 mg/kg DRPH

0.092 mg/kg E

0.375 mg/kg X

EFFLUENT OUTFALL

SEWAGE  
TREATMENT  
UNIT

INTERCEPTION  
TRENCH

3S26-5

11800 mg/kg DRPH

0.949 mg/kg T

4.53 mg/kg E

4.23 mg/kg X

3S27-2.5

228 mg/kg DRPH

ND BTEX

2S13-1.5

0.2 ppm VOC

ND DRPH

ND RRPH

BERM

2S20

490 ppm DRPH

ROAD

S02-2.5

2300 ppm DRPH

13 ppm GRPH

ND RRPH

ND BTEX

TANK 1

3S31-5

3130 mg/kg DRPH

0.113 mg/kg T

2.66 mg/kg E

4.04 mg/kg X

S03-5.5


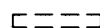









4500 ppm DRPH

25 ppm GRPH

ND RRPH

ND BTEX

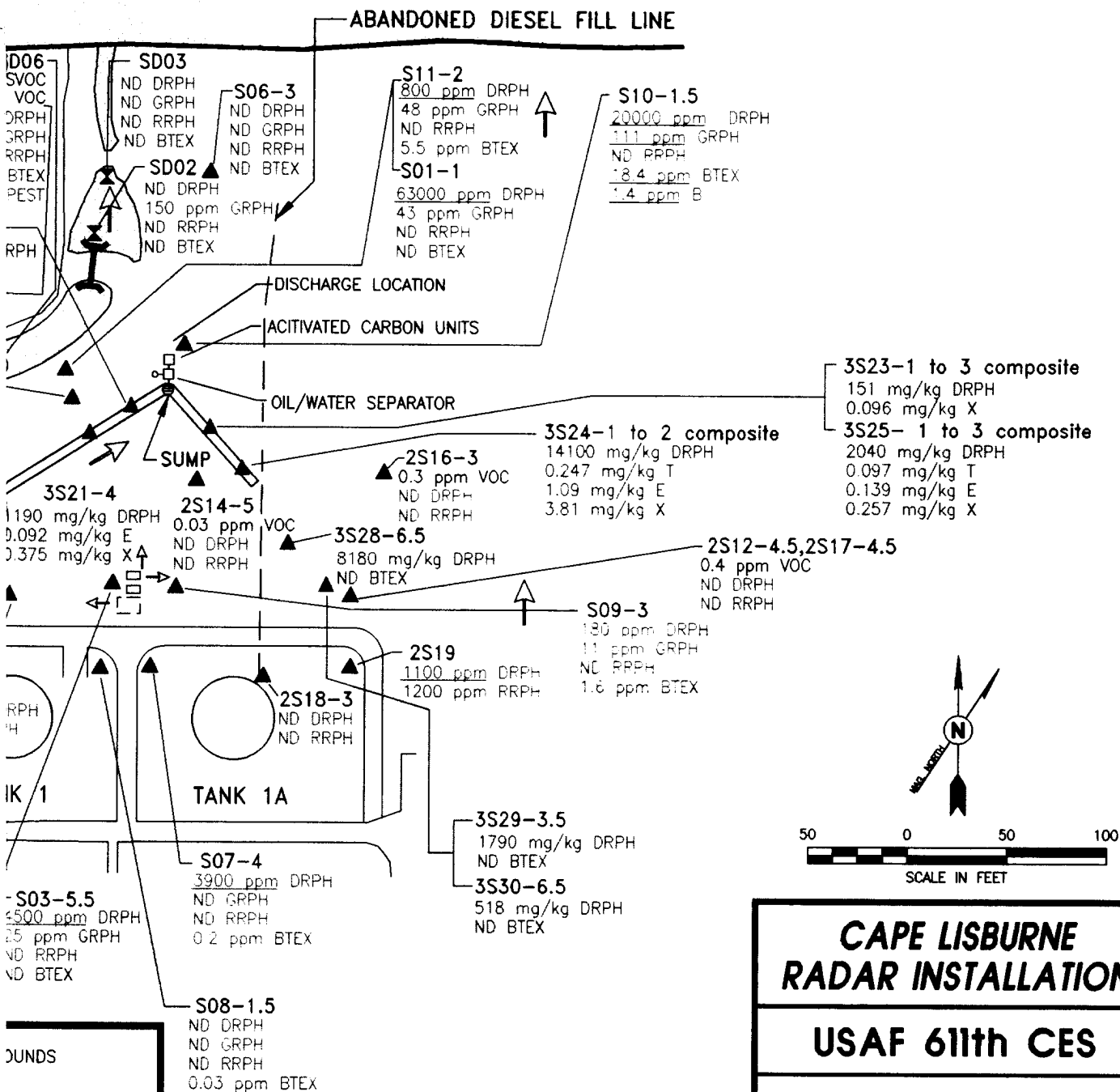
# LEGEND

-  BUILDINGS, STRUCTURES
-  ABANDONED BUILDINGS, STRUCTURES
-  ROADS
-  SOIL SAMPLE
-  SEDIMENT SAMPLE
-  SURFACE WATER SAMPLE
-  SEDIMENT AND WATER SAMPLES
- 2.6 ppm 1993 CT&E DATA
- 0.9 ppm F&B DATA
- 2.6 ppm 1994 CT&E DATA
-  SURFACE WATER
-  CULVERT
-  GRAVEL PAD BOUNDARY
-  SURFACE DRAINAGE

0000 CONCENTRATIONS ARE ABOVE ACTION LEVELS

- ND NO CONTAMINATION DETECTED
- SVOC TOTAL SEMI-VOLATILE ORGANIC COMPOUNDS
- VOC TOTAL VOLATILE ORGANIC COMPOUNDS
- DRPH DIESEL RANGE PETROLEUM HYDROCARBONS
- GRPH GASOLINE RANGE PETROLEUM HYDROCARBONS
- RRPH RESIDUAL RANGE PETROLEUM HYDROCARBONS

- BTEX TOTAL BTEX COMPOUNDS
- B BENZENE
- T TOLUENE
- E ETHYLBENZENE
- NPH NAPHTHALENE
- 1,2,4 1,2,4-TRIMETHYLBENZENE
- 1,3,5 1,3,5-TRIMETHYLBENZENE
- X TOTAL XYLENES



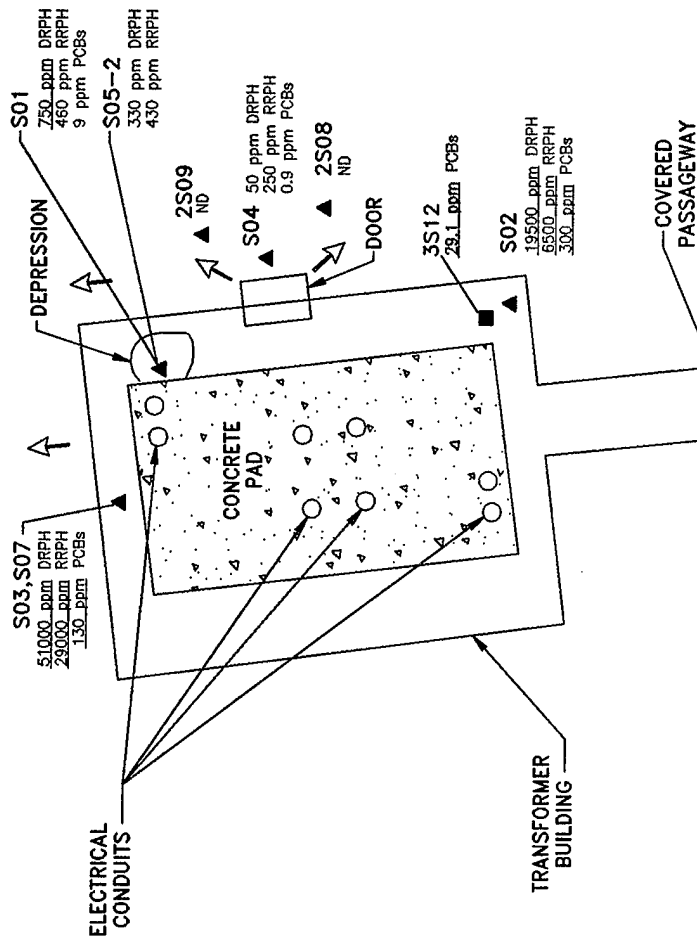
## CAPE LISBURN RADAR INSTALLATION

### USAF 611th CES

FIGURE NO. 2-5

SPILL/LEAK #3 (ST07)  
SAMPLE LOCATIONS  
AND  
ANALYTICAL RESULTS

DRAWING No. 94SS08A



# CAPE LISBURNERADAR INSTALLATION

## USAF 611th CES

FIGURE NO. 2-6  
UPPER CAMP TRANSFORMER  
BUILDING (SS08)  
SAMPLE LOCATIONS  
AND  
ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK



exceeded the ARARs for petroleum hydrocarbon contamination of soil (ADEC 1991). Aroclor 1254 and Aroclor 1260 exceeded the RBSLs based on cancer risk and noncancer hazard and the ARAR for PCBs under the TSCA.

**Surface Water.** No surface water bodies were identified at the Upper Camp Transformer Building (SS08); therefore, no surface water COCs have been identified (Figure 2-6, Table 2-1).

#### **2.1.5.5 Lower Camp Transformer Buildings (SS09).**

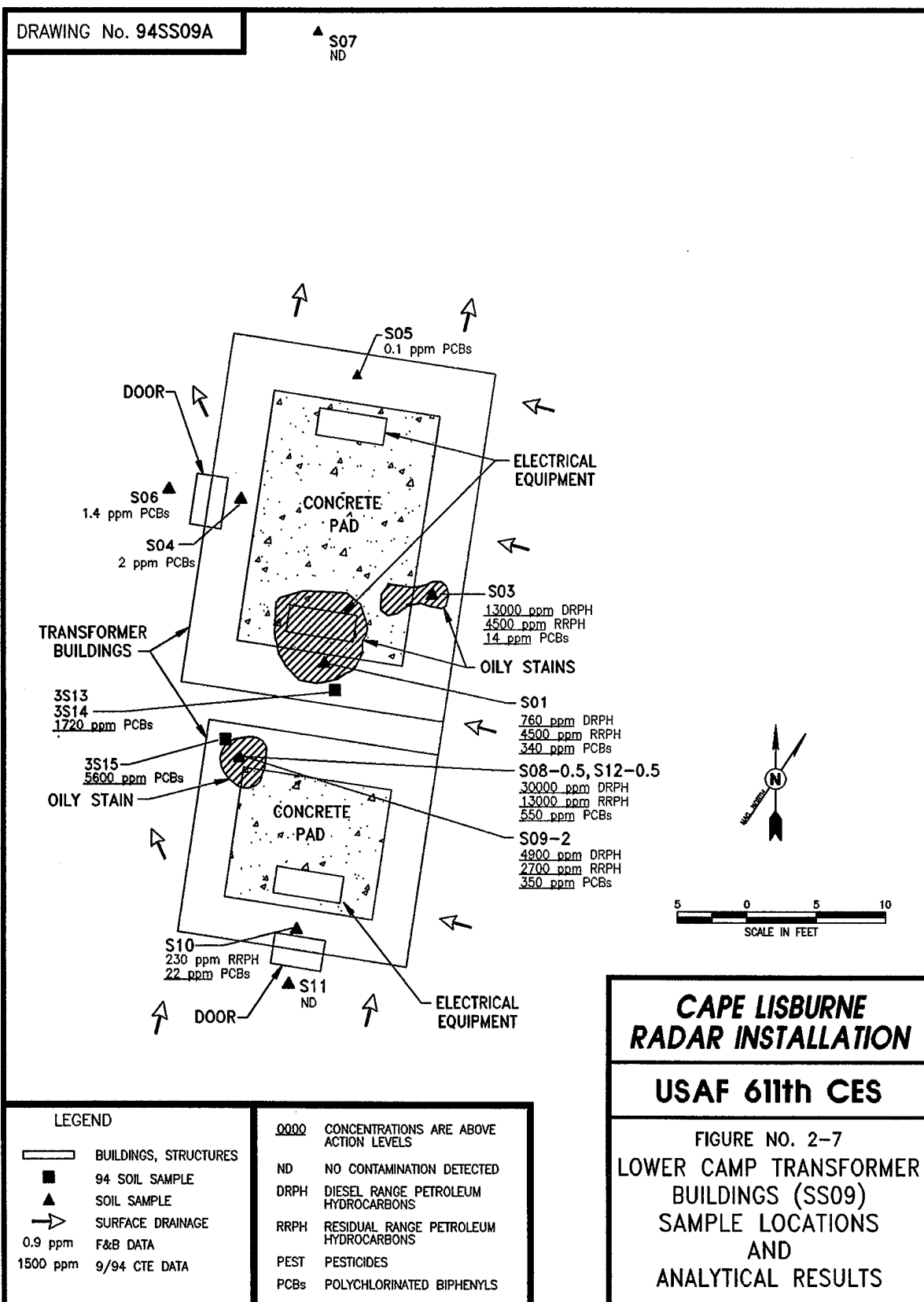
**Soil or Sediment.** DRPH, RRPH, Aroclor 1254, and Aroclor 1260 were identified as COCs for the soil matrix at the Lower Camp Transformer Buildings (SS09) (Figure 2-7, Table 2-1). The maximum concentrations of DRPH and RRPH exceeded their background concentrations and the ARAR concentrations for petroleum hydrocarbon contamination of soil (ADEC 1991). The maximum concentrations of the Aroclors exceeded the RBSLs based on cancer risk and noncancer hazard and the ARAR for PCBs under the TSCA.

**Surface Water.** No surface water bodies were identified at the Lower Camp Transformer Buildings (SS09); therefore, no surface water COCs have been identified (Figure 2-7, Table 2-1).

**2.1.5.6 Summary of Chemicals of Concern.** The assessment of human health risk at the Cape Lisburne radar installation is based on the COCs identified in this section. A summary of the COCs is presented in Table 2-3.

THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. 94SS09A



THIS PAGE INTENTIONALLY LEFT BLANK

## 2.2 EXPOSURE ASSESSMENT

The exposure assessment section of a baseline human health risk assessment identifies and describes potential receptors and the exposure pathways by which exposure may occur, and estimates the magnitude of those exposures. This section includes an analysis of which pathways are complete (Section 2.2.1), migration and fate of COCs (Section 2.2.2), an estimation of the total intake of the chemicals (Section 2.2.3), and a summary of how the average daily dose (ADD) was calculated (Section 2.2.4).

### 2.2.1 Pathway Analysis

Pathway analysis involves the evaluation of the components of potential exposure pathways and a determination of whether each pathway is complete. An exposure pathway describes the course a chemical will take from a source to an exposure point where a receptor can come into contact with it. A complete exposure pathway has five components:

- source of contamination;
- release mechanism;
- transport mechanism;
- exposure point; and
- receptor.

If one component of an exposure pathway is missing, then exposure will not occur, and there is no health risk. For example, if a shallow aquifer was contaminated with tetrachloroethene but that aquifer was not used as a water supply, no exposure point would exist and a ground water ingestion pathway would not be complete.

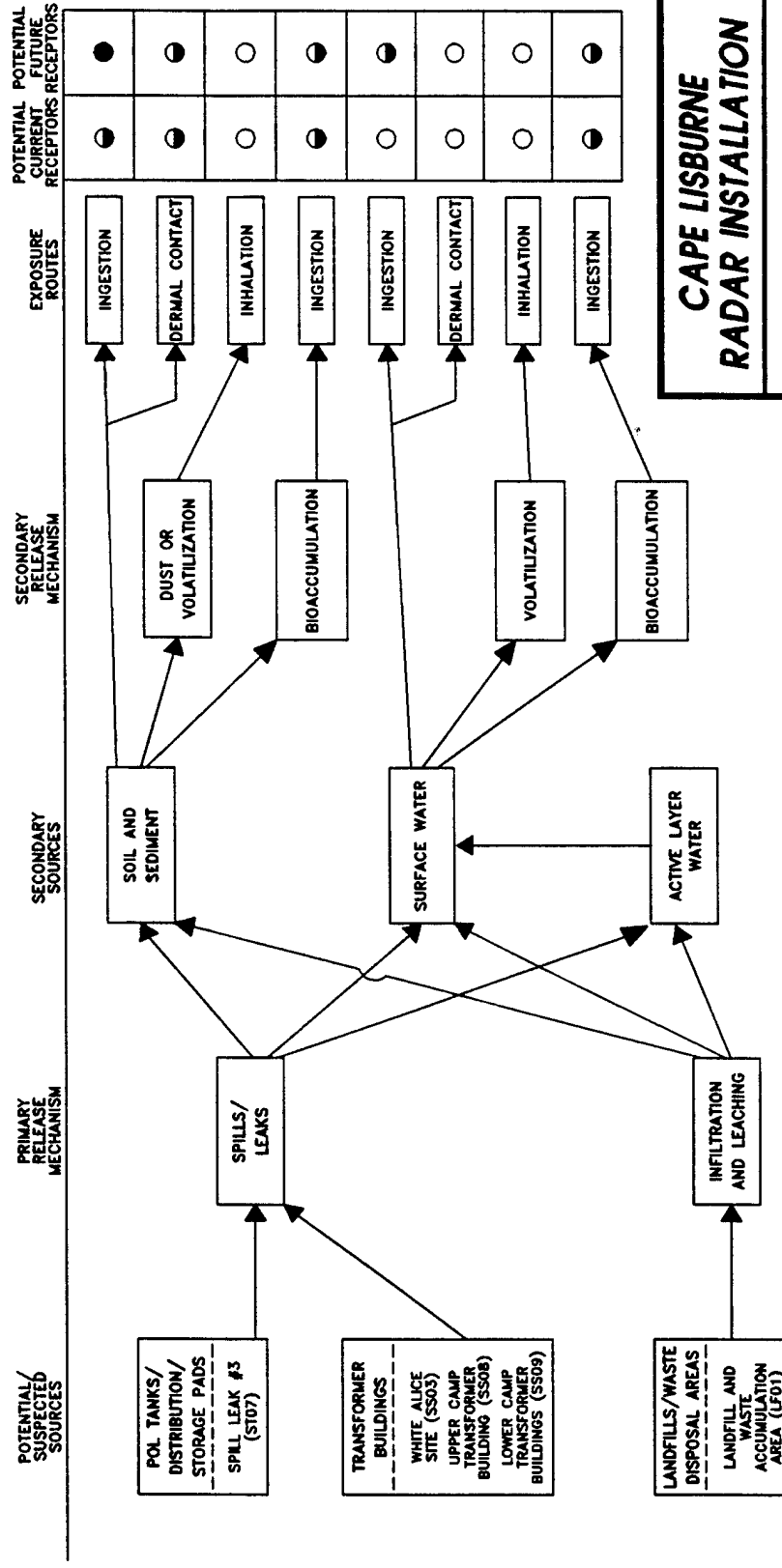
The potential exposure pathways evaluated for the Cape Lisburne human health risk assessment are presented in Figure 2-8 and Table 2-4, and are discussed in Sections 2.2.1.1 through 2.2.1.4.

**2.2.1.1 Soil and Sediment Ingestion.** Cape Lisburne installation workers and residents of the North Slope Borough may be exposed to soil and sediment contaminated by previous operations at the installation. The most likely exposure routes are incidental ingestion of soil and dermal absorption of contaminants in the soil. Site-specific characteristics will limit the magnitude, frequency, and duration of exposures to soil and sediment. The ground is covered with snow and ice, which eliminate soil or sediment exposure, for approximately nine months of the year. In the summer months when snow cover is generally absent, cool temperatures (30°F to 46°F) (University of Alaska 1978), keep both workers and villagers in heavy, long-sleeved clothing and gloves that eliminate dermal contact with and hand-to-mouth transfer of soil. Therefore, although both the incidental soil ingestion and dermal contact pathways are considered to be potentially complete, only incidental ingestion of soil or sediment will be evaluated further in this risk assessment.

The exposure assumptions used to evaluate the soil and sediment ingestion pathway are upper-bound residential scenario assumptions and, therefore, probably overestimate the true hazard or risk associated with this pathway. The purpose of using residential assumptions is to evaluate

THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. LIS-FLOW



**CAPE LISBURN  
RADAR INSTALLATION**

**USAF 611th CES**

FIGURE NO. 2-8

HUMAN HEALTH  
RISK ASSESSMENT  
POTENTIAL EXPOSURE  
PATHWAYS

- COMPLETE EXPOSURE PATHWAY FOR HUMANS (DEWLINE WORKERS AND NATIVE NORTHERNERS)
- ◐ POTENTIALLY COMPLETE PATHWAY
- INCOMPLETE PATHWAY

THIS PAGE INTENTIONALLY LEFT BLANK



**TABLE 2-4. EXPOSURE PATHWAY ANALYSIS FOR CAPE LISBURNE HUMAN HEALTH RISK ASSESSMENT**

POTENTIALLY CONTAMINATED MEDIUM	POTENTIAL ROUTES OF EXPOSURE	POTENTIAL RECEPTORS	PATHWAY COMPLETE?	EXPOSED POPULATION ESTIMATE
Soil	Ingestion, dermal absorption	Radar installation workers, North Slope residents.	Ingestion, Yes Dermal Contact, No	100 <sup>a</sup>
Sediments	Ingestion, dermal absorption	Radar installation workers, North Slope residents.	Ingestion, Yes Dermal Contact, No	100 <sup>a</sup>
Air	Inhalation of volatiles from soil or surface water or inhalation of fugitive dust	Radar installation workers, North Slope residents.	No, volatile concentrations in soil and surface water are very low; dust generation is not likely due to marshy vegetated landscape and high humidity.	0
Surface Water	Incidental ingestion, dermal absorption	Radar installation workers, North Slope residents.	Maybe, drinking water supplies are either upgradient from installation or in unaffected areas; fishing occurs in unaffected areas; swimming does not occur onsite; however, incidental exposure may occur during installation operations or trespassing by native villagers.	100 <sup>a</sup>
Ground Water	Ingestion, dermal absorption	Radar installation workers, North Slope residents.	No, permafrost limits presence of ground water to shallow active layer. Ground water is not used for any purpose. Water for domestic use is obtained from the Water Gallery (AOC3) which is upgradient of areas that may have been impacted by the installation. Sampling at the Water Gallery has confirmed the absence of any impact on the installation water supply.	0

<sup>a</sup> Assumes future residential land use. Currently there are no residents other than the workers stationed at Cape Lisburne. The nearest village is Point Hope, approximately 35 miles to the southwest. The exposed population of 100 is an estimate of the size of a village that might exist at Cape Lisburne if the installation was ever released for residential use.

the hazard or risk associated with future residential use of the Cape Lisburne installation. Although the Air Force does not currently plan to deactivate the installation, it is possible that the installation may be retired and released for civilian use, at which time residential use of the installation may occur.

**2.2.1.2 Inhalation.** Cape Lisburne installation workers and native northerners may be exposed to site contamination by inhalation of organic compounds volatilized from the soil or surface water, or windborne dust to which contamination has adsorbed. These exposure pathways are not considered complete for the Cape Lisburne risk assessment because snow and ice cover the site for approximately nine months of the year and, during the summer months, the high humidity, vegetative cover, and thawing of surface and active layer water significantly limit the entrainment of dust particles in ambient air. The generally low temperatures and high moisture content of the soil also tend to inhibit volatilization. The inhalation pathway will not be considered further in this risk assessment.

**2.2.1.3 Water Ingestion.** Surface water features, particularly those potentially affected by operations at the installation, are not likely to be used for drinking or other domestic purposes even on an incidental basis. This is because these surface water features are not reliable, being frozen for most of the year. Ingestion of surface water will, however, be considered a potentially complete exposure pathway under a future use scenario. Under current conditions, the Water Gallery supplies the domestic and industrial water demands of the installation. Analyses of water samples from the Water Gallery indicate that no contaminants are present. In addition, no known areas of contamination exist upgradient of the Water Gallery.

**2.2.1.4 Ground Water.** Permafrost limits the presence of ground water to the active layer which thaws during the summer months. The water present in the active layer is not known to be used for any purpose, with the exception of the Water Gallery System. Water for domestic use is obtained from the Water Gallery (AOC3) which is upgradient of areas that may have been impacted by the installation. Sampling at the Water Gallery has confirmed the absence of any impact on the installation water supply. Therefore, the ground water pathway will be eliminated from consideration in this risk assessment.

## **2.2.2 Migration and Fate of Chemicals of Concern**

The COCs selected for Cape Lisburne generally fall into four classes:

- Refined and residual petroleum hydrocarbons (DRPH, GRPH, and RRPH);
- Volatile organic compounds (VOCs: benzene, carbon tetrachloride, chloroform, tetrachloroethene, and trichloroethene);
- Metals (barium and manganese); and
- Polychlorinated biphenyls (PCBs: Aroclor 1254 and 1260).

This section presents a summary of the migration and fate of each of these classes given the environmental conditions at Cape Lisburne.

Once released to the environment, the COCs are immediately subject to several processes, including evaporation and volatilization, bulk flow, soil adsorption, dissolution in surface or active layer water, biodegradation, and photooxidation. The extent to which the COCs undergo each of these processes depends on their chemical and physical properties (e.g.,  $K_{oc}$ ,  $K_{ow}$ , water solubility, vapor pressure, Henry's law constant), the volume released, soil flora, meteorological conditions, and soil and water organic carbon content.

The migration of petroleum hydrocarbons released to the gravel pads and tundra is expected to follow the rank order: GRPH > DRPH > RRPB. GRPH is generally considered to include hydrocarbons with carbon chain ranges from C5 to C12 that tend to be relatively mobile and less persistent than longer chain hydrocarbons. Depending on the length of time since a spill or leak occurred, the petroleum hydrocarbons observed in soil samples would be expected to be enriched in components that have carbon chain ranges greater than C10 or C11, high  $K_{oc}$ , and  $K_{ow}$  values, and low vapor pressure and water solubility, and are not rapidly biodegradable. Petroleum components that fit this profile are higher molecular weight n-alkanes, mono- and polyaromatics, and cycloalkanes. These components would tend to appear in laboratory analyses as diesel range or heavy oil range organics (DRPH and RRPB).

The migration of VOCs is expected to be rapid compared to the petroleum hydrocarbons. These compounds tend to have high vapor pressures which favor volatilization, high water solubility, and low  $K_{oc}$  and  $K_{ow}$  values. Therefore, the VOCs would tend to be highly mobile in the environment and dissipate rapidly after a spill or leak. In the results of field sampling, VOC concentrations would be expected to be fairly low depending on the time since the spill or leak occurred. The frigid conditions on the North Slope, however, would tend to reduce the mobility due to volatilization or evaporation.

The metals observed at Cape Lisburne are probably of natural origin and not due to the operation of, or activities at, the radar installation. The presence of barium and manganese in surface water samples is most often associated with landfill leachate since the anaerobic and acidic conditions inside of a landfill tends to release naturally occurring metals from the soil. Metals will tend to be persistent and of low mobility in the environment.

PCBs also have a low migratory potential. These compounds are generally of high molecular weight, low vapor pressure, and water solubility, and high  $K_{oc}$  and  $K_{ow}$  values. Biodegradation of PCBs on the North Slope would also be expected to be minimal given the frigid conditions and presence of permafrost.

In conclusion, the COCs observed at the Cape Lisburne installation are generally expected to be fairly persistent and of low mobility. Exposure by contact with soils, primarily through accidental ingestion, is expected to predominate compared to exposure by inhalation.

### 2.2.3 Estimation of Chemical Intake

The exposure assessment for the Cape Lisburne radar installation required the development of site-specific assumptions because of the unique location (no nearby villages) and limited workers stationed at the installation. This section of the report is focused on the exposure variables for which site-specific assumptions were made. These variables include:

- exposure frequency;
- exposure duration;
- ingestion of locally produced meat (e.g., caribou, fish, and birds);
- ingestion of locally produced vegetation (e.g., berries);
- soil ingestion rate; and
- rate of dermal contact with soil.

The exposure assumptions used in the human health risk assessment are presented in Table 2-5.

Three potential receptor groups will be evaluated for the Cape Lisburne risk assessment: an adult assigned to maintenance work at the Cape Lisburne installation (radar installation worker), an adult native of the North Slope of Alaska (native), and a native child (child). The native adult and child are considered to represent the reasonable maximum exposure that might occur at the

**TABLE 2-5. EXPOSURE ASSUMPTIONS FOR ESTIMATING CHEMICAL INTAKE**

PARAMETER	RADAR INSTALLATION WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN CHILD
Exposure Frequency - Soil Ingestion (days/year)	30	30	30
Exposure Frequency - Water Ingestion (days/year)	180	180	N/A
Exposure Duration (years)	10	55 <sup>a</sup>	6 <sup>a</sup>
Soil Ingestion Rate (mg/day)	50	100	200
Drinking Water Ingestion Rate (L/day)	2	2	N/A
Average Body Weight (kg)	70	70	15
Averaging Time (days)	25,550 (cancer) 3,650 (noncancer)	25,550 <sup>b</sup> (cancer) 20,075 <sup>c</sup> (noncancer) 17,885 <sup>d</sup> (noncancer)	2,190 <sup>d</sup> (noncancer)

N/A Not applicable; drinking water pathway evaluated for adult only.

<sup>a</sup> Exposure duration for water ingestion pathway is 55 years. For soil ingestion, exposure duration is 6 years as a child and 49 years as an adult.

<sup>b</sup> Averaging time for the evaluation of cancer risk by the soil and water ingestion pathways.

<sup>c</sup> Averaging time for the evaluation of noncancer hazard by the water ingestion pathway.

<sup>d</sup> Averaging time for the evaluation of noncancer hazard by the soil ingestion pathway.

installation under a future use scenario that includes residential receptors. Although there are no plans to do so, the Cape Lisburne installation may be released for civilian residential use in the future.

The estimation of chemical intake requires the evaluation of several exposure variables: exposure point concentration; exposure frequency; exposure duration; ingestion of locally produced meat, fish, and vegetation; soil ingestion; drinking water ingestion; dermal contact with soil; inhalation; and body weight. These exposure variables are discussed in the following sections.

**2.2.3.1 Exposure Point Concentration.** Based on the amount of analytical data available for the risk assessment of the Cape Lisburne installation, and the requirement that the risk characterization be conducted individually for each of the five sites, only maximum concentrations of the COCs were used for exposure point concentrations. This approach yields a conservative upper-bound estimate of the ADD to which potential receptors may be exposed.

**2.2.3.2 Exposure Frequency.** The exposure frequency variable is an estimate of the amount of time a potential receptor may come in to contact with contaminated media. For the radar installation worker, the exposure frequency estimate is based on a duty rotation of eleven months on-site, one month off-site. These estimates are based on knowledge of the site worker's daily activities and vacation allowance. During the eleven months of on-site duty, it is estimated that the worker is outside for four hours per day. The remaining 20 hours is spent inside the module trains or enclosed vehicles, where exposure to contaminated media is not expected to occur. An estimated exposure frequency for the radar installation worker is, therefore, 11 months/year x 30 days/month x 4 hours/day x 1 day/24 hours = 55 days/year. The primary environmental medium of concern is, however, contaminated soil, and this estimate of exposure frequency does not account for the number of days per year that snow covers the ground and eliminates the potential for contact with contaminated soil. Six months is a conservative average estimate of the number of months per year of snow cover at all eight radar installation installations. To be even more conservative, it is assumed that a worker's tour of duty includes all six of the months without snow cover, thus the soil ingestion exposure frequency of 30 days/year for the radar installation worker is recommended.

The soil ingestion exposure frequency estimate for a native adult or child of the North Slope is based on an estimate of the frequency with which the individual would be at a radar installation involved in activities that include exposure to soil (assuming a potential future residential scenario). In this case, a conservative estimate of exposure would be 4 hrs/day x 30 days per month x 1 day/24 hrs x 6 months of exposed soil = 30 days per year.

The exposure frequency for water ingestion was conservatively estimated at 180 days/year that surface water would be available (i.e., not frozen), and is based on a potential future scenario where the contaminated site surface water is used as drinking water source.

**2.2.3.3 Exposure Duration.** The exposure duration variable is an estimate of the amount of time a potential receptor will remain at or near a radar installation over a lifetime. For the radar installation worker the exposure duration is an estimate of the maximum tour of duty at an

installation. A conservative estimate of the duration of a tour at a particular installation is 10 years. For the potential native receptor, a conservative estimate of exposure duration is 55 years based on the assumption that the site use will be residential. EPA's default reasonable maximum exposure duration is 30 years; however, this is based on the overall U.S. population. Because the Alaskan natives are more likely to remain in their village for a longer period, 55 years was determined to be a more appropriate estimate based on best professional judgement.

**2.2.3.4 Averaging Time.** The averaging time represents the period of time over which exposure is averaged and is based on the assumption that intermittent exposure at a given contaminant concentration is equivalent to a continuous exposure at a lower concentration. For the radar installation worker, the averaging time is based on the EPA default lifetime of 70 years for evaluation of carcinogens, and 10 years (equivalent to the exposure duration) for the evaluation of noncarcinogens. For the native northern adult receptors an averaging time of 70 years for carcinogens was also chosen. To evaluate exposure to noncarcinogens in soil and sediment for the native northern adult and child, an averaging time of 49 years as an adult and 6 years as a child was used (to account for 55 year total exposure). To evaluate the exposure of native northern receptors to noncarcinogens in water an averaging time of 55 years was used.

**2.2.3.5 Ingestion of Locally Produced Meat, Fish, and Vegetation.** The food supplies of radar installation workers are largely imported from outside the area. Occasionally, a worker would be expected to ingest a locally caught fish or game animal, but the frequency and magnitude of this ingestion is expected to have a negligible effect on exposure to the COCs. Food supplies for the residents of the North Slope Borough are partly imported from outside the area, and some reports indicate that the reliance on hunting and fishing for subsistence is decreasing as the economy moves from subsistence to wage labor (Chance 1990). Although Inupiat in general have less time to hunt and fish than in the past, up to 50 percent of their food may derive from subsistence activities and this level may be increasing (Harcharek 1994). Most of the hunting and fishing occurs away from the Cape Lisburne radar installation in areas unaffected by the installation. It is not likely that contamination observed at the installation has affected the mammals, birds, and vegetation that may be collected for consumption on the North Slope. Therefore, the consumption of locally produced food is not likely to pose a significant risk of adverse health effects and will not be considered a complete exposure pathway. The ERA, Section 3.0, presents a detailed assessment of risks to ecological receptors.

**2.2.3.6 Soil Ingestion Rate.** A conservative approach to estimating soil ingestion rate is to assume that the EPA default soil ingestion rates of 50 mg/day for workers (EPA 1991a) and 100 mg/day for adults in a residential setting (EPA 1989a). The EPA default soil ingestion rate for children is 200 mg/day (EPA 1989a, 1991a); this is the recommended value for the risk assessment.

**2.2.3.7 Drinking Water Ingestion Rate.** There are no circumstances at the Cape Lisburne installation that would invalidate the EPA default adult drinking water ingestion rate of 2 L/day. Therefore, this is the recommended value for both workers and natives. However, drinking water is obtained from the Water Gallery. Sampling at the Water Gallery has confirmed the absence of any impact on the installation water supply, so this is a route of potential exposure.

By convention (EPA 1989a), noncancer hazard and cancer risk associated with the drinking water pathway are evaluated for an adult receptor, not a child (Table 2-5). The basis for this approach is that the ratio of drinking water ingestion rate to body weight is assumed to remain relatively constant from childhood to adulthood.

**2.2.3.8 Dermal Contact with Soil Rate.** Because of the harsh North Slope weather, potential receptors (both workers and natives) are expected to be heavily clothed and gloved. Observations made by RI field personnel indicate that potential human receptors were heavily clothed during the months of the field investigations (August and September 1993). Therefore, dermal exposure to contaminated soils is considered negligible. In addition, the duties of installation workers that involve soil work (excavating, grading, etc.) are normally conducted in equipment with enclosed cabs. Thus, a dermal contact rate does not appear to be necessary for the exposure assessment.

**2.2.3.9 Inhalation Rate.** The inhalation pathway is not complete (Section 2.2.1.2), so no estimate for this variable is necessary.

**2.2.3.10 Body Weight.** There are no circumstances at the Cape Lisburne installation that would invalidate the EPA default adult body weight of 70 kg. Therefore, this is the recommended value for both workers and natives. The recommended body weight for children is the EPA default value of 15 kg.

## **2.2.4 Quantifying Exposure**

For each complete, or potentially complete, exposure pathway at the Cape Lisburne installation (soils ingestion, drinking water ingestion), the ADD for estimating noncancer hazard and the lifetime average daily dose (LADD) for estimating excess lifetime cancer risk were calculated. The equations used for the calculation of ADD and LADD are presented in Table 2-6.

The exposure assumptions assigned to each variable in these equations are presented in Table 2-4. The estimates of ADD and LADD for the COCs at each site are presented in the risk characterization spreadsheets in Appendix A.

TABLE 2-6. EQUATIONS USED FOR ESTIMATING POTENTIAL DOSE

EXPOSURE ROUTE	EQUATION	PARAMETER DEFINITIONS
Ingestion of Soil	<p>Native Northern Adults/Children</p> $ADD \text{ or } LADD \text{ (mg/kg/day)} = \frac{C_s * CF * EF}{AT} \sum_{i=1}^n \frac{IR_i * ED_i}{BW_i}$ <p>Radar installation workers:</p> $ADD \text{ or } LADD \text{ (mg/kg/day)} = \frac{C_s * CF * IR * EF * ED}{BW * AT}$	<p><math>C_s</math> = concentration in soil (mg/kg)</p> <p><math>CF</math> = conversion factor (<math>10^{-6}</math> kg/mg)</p> <p><math>IR</math> = ingestion rate (mg/day)</p> <p><math>EF</math> = exposure frequency (days/year)</p> <p><math>ED</math> = exposure duration (years)</p> <p><math>BW</math> = body weight (kg)</p> <p><math>AT</math> = averaging time (days/year x years)</p>
Ingestion of Surface Water	$ADD \text{ or } LADD \text{ (mg/kg/day)} = \frac{C_w * CF * IR * EF * ED}{BW * AT}$	<p><math>C_w</math> = concentration in surface water (µg/L)</p> <p><math>CF</math> = conversion facton (<math>10^{-3}</math> mg/µg)</p> <p><math>IR</math> = ingestion rate (L/day)</p> <p><math>EF</math> = exposure frequency (days/year)</p> <p><math>ED</math> = exposure duration (years)</p> <p><math>BW</math> = body weight (kg)</p> <p><math>AT</math> = averaging time (days/year x years)</p>



## 2.3 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to weigh available evidence regarding the potential for particular contaminants to cause adverse effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a contaminant and the increased likelihood or severity of adverse effects or both. This is done separately for noncarcinogenic effects (Section 2.3.1) and carcinogenic effects (Section 2.3.2). Toxicity summaries are presented in Section 2.3.3.

Toxicity assessment for environmental contaminants is generally accomplished in two steps: hazard identification and dose-response assessment. Hazard identification is the process of determining whether exposure to an agent can cause an increase in the incidence of a particular adverse health effect (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans. Hazard identification involves characterizing the nature and strength of the evidence of causation. Dose-response evaluation is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose of the contaminant administered or received and the incidence of adverse health effects in the exposed population. From this quantitative dose-response relationship, toxicity values [e.g., RfDs and slope factors (SFs)] are derived that can be used to estimate the incidence or potential for adverse effects as a function of human exposure to the agent. These toxicity values are used in the risk characterization step to estimate the likelihood of adverse effects occurring in humans at particular exposure levels.

### 2.3.1 Toxicity Assessment for Noncarcinogenic Effects

A reference dose, or RfD, is the toxicity value used most often in evaluating noncarcinogenic effects resulting from exposures at contaminated sites. Various types of RfDs are available depending on the exposure route (oral or inhalation), the critical effect (developmental or other), and the length of exposure being evaluated (chronic, subchronic, or single event). The oral RfDs used to estimate the noncancer hazard associated with exposure to soils, sediments, and surface water at the Cape Lisburne facility are presented in Table 2-7.

A chronic RfD is defined as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound. Chronic RfDs generally should be used to evaluate the potential noncancerous effects associated with exposure periods between 7 years (approximately 10 percent of a human lifetime) and a lifetime. Many chronic RfDs have been reviewed and verified by an intra-Agency RfD Workgroup and entered into EPA's IRIS database.

**2.3.1.1 Concept of Threshold.** For many noncancerous effects, protective mechanisms are believed to exist. They must be overcome before the adverse effect is manifested. For example, where a large number of cells perform the same or similar function, the cell population may have to be significantly depleted before the adverse effect is seen. As a result, a range of

TABLE 2-7. TOXICITY CRITERIA FOR NONCANCER EFFECTS OF THE CHEMICALS OF CONCERN FOR CAPE LISBURNE

CHEMICAL	ORAL REFERENCE DOSE (RfD) (mg/kg-day)	TARGET ORGAN OR CRITICAL EFFECT (species) <sup>a</sup>	UNCERTAINTY FACTOR <sup>b</sup>	ORAL RfD SOURCE <sup>c</sup>
DRPH	0.08 <sup>d</sup>	liver effects (mice)	10,000	ECAO
GRPH	0.2 <sup>d</sup>	decreased body weight (rats)	1,000	ECAO
RRPH	0.08 <sup>d</sup>	decreased body weight (rats)	1,000	ECAO
Aroclor 1254	0.00002	ocular exudate, inflamed Meibomian glands, distorted nail growth, immunosuppression (monkey)	300	IRIS
Aroclor 1260	0.00002 <sup>e</sup>	NA	NA	NA
Benzene	NA	NA	NA	IRIS
Carbon tetrachloride	0.0007	liver lesions	1,000	IRIS
Chloroform	0.11	fatty cyst formation in liver	1,000	IRIS
Tetrachlorethene	0.01	liver toxicity	1,000	IRIS
Trichloroethene	NA	NA	NA	IRIS
Barium	0.07	NOEL for increased blood pressure	3	IRIS
Manganese (water)	0.005	CNS effects	1	IRIS
Manganese (soil)	0.14	CNS effects	1	IRIS

<sup>a</sup> A target organ is the organ apparently most sensitive to the toxicity of a chemical. A critical effect is reported when EPA has not identified a target organ for the toxicity of a given chemical.

<sup>b</sup> The uncertainty factors used to develop oral reference doses are generally applied in multiples of 10 to account for shortcomings in the toxicological database. The greater the uncertainty factor, the lower the confidence level in the RfD. A factor of 10 is applied to account for each of the following: human variability in toxic response, extrapolation from animal studies to humans, extrapolation of short-term exposures to long-term exposures, and for the extrapolation of a lowest-observed adverse effect level (LOAEL) to a no observed adverse effect level (NOAEL).

<sup>c</sup> Sources of oral RfD values are IRIS, HEAST, or ECAO.

<sup>d</sup> Oral RfD values for DRPH, GRPH, and RRPH are based on (EPA 1992b) and are considered provisional RfDs.

<sup>e</sup> This oral reference dose for Aroclor 1260 has been extrapolated from the oral reference dose for Aroclor 1254.

NA  
Not available.

exposures from zero to some finite level exists that can be tolerated by the organism with essentially no chance of expression of adverse effects. In developing a toxicity value for evaluating noncancerous effects (i.e., an RfD), the approach is to identify the upper bound of this tolerance range (i.e., the maximum subthreshold level). Because variability exists among humans, attempts are made to identify a subthreshold level that protects sensitive individuals in the population. For most chemicals, this level can only be estimated; the RfD incorporates uncertainty factors indicating the degree of extrapolation used to derive the estimated value. RfD summaries in IRIS also contain a statement expressing the overall confidence that the evaluators have in the RfD (high, medium, or low). The RfD is generally considered to have uncertainty spanning an order of magnitude or more, so the RfD should not be viewed as a strict scientific demarcation between levels that are toxic and nontoxic.

### **2.3.2 Toxicity Assessment for Carcinogenic Effects**

An SF and the accompanying weight-of-evidence determination are the toxicity data most commonly used to evaluate potential human carcinogenic risks. The methods EPA uses to derive these values are outlined below. Additional information can be obtained by consulting EPA's *Guidelines for Carcinogen Risk Assessment* (EPA 1986a) and IRIS Background Document #2 (IRIS 1994). The SFs for the COCs at Cape Lisburne are presented in Table 2-8.

**2.3.2.1 Concept of Nonthreshold Effects.** Risk evaluation based on the presumption of a dose-response threshold is generally thought to be inappropriate for carcinogenesis. In the evaluation of carcinogens, EPA assumes that a small number of molecular events can evoke changes in a single cell and lead to uncontrolled cellular proliferation and eventually to clinical state of disease (cancer). This hypothesized mechanisms for carcinogenesis is referred to as "nonthreshold" because all levels of exposure pose some probability of generating a carcinogenic response. Thus, no dose is thought to be risk-free, and an effect threshold cannot be estimated.

For carcinogenic effects, EPA uses a two-part evaluation in which the substance first is assigned a weight-of-evidence classification, then a SF is calculated.

**2.3.2.2 Assigning a Weight-of-Evidence.** In the first step of the evaluation, the carcinogenicity data are evaluated to determine the likelihood that the agent is a human carcinogen. The evidence is characterized separately for human studies and animal studies as sufficient, limited, inadequate, no data, or evidence of no effect. The characterizations of these two types of data are combined and, based on the extent to which the agent has been shown to be a carcinogen in experimental animals, humans, or both, the agent is given a provisional weight-of-evidence classification. EPA scientists then adjust the provisional classification upward or downward based on other supporting evidence of carcinogenicity.

The EPA classification system for weight-of-evidence is shown in Table 2-9.

**2.3.2.3 Generating a Slope Factor.** For chemicals classified as known or probable human carcinogens, a toxicity value that defines quantitatively the relationship between dose and response (i.e., the SF) is calculated. SFs are typically calculated for potential carcinogens in

TABLE 2-8. TOXICITY VALUES FOR THE CARCINOGENICITY OF THE CHEMICALS OF CONCERN AT CAPE LISBURNE

CHEMICAL	WEIGHT-OF-EVIDENCE (WOE)	TUMOR TYPE (species)	ORAL SLOPE FACTOR (kg-day/mg)	ORAL SLOPE FACTOR SOURCE <sup>a</sup>
DRPH	NA	NA	NA	NA
GRPH	C	liver adenoma/carcinoma (mouse)	0.0017	ECAO
RRPH	NA	NA	NA	NA
Aroclor 1254	B2	trabecular carcinoma/adenocarcinoma, neoplastic nodule	7.7	IRIS
Aroclor 1260	B2 <sup>b</sup>	trabecular carcinoma/adenocarcinoma, neoplastic nodule	7.7	IRIS
Benzene	A	leukemia (human)	0.029	IRIS
Carbon tetrachloride	B2	hepatocellular carcinomas, hepatomas (various)	0.13	IRIS
Chloroform	B2	kidney tumors (rat)	0.0061	IRIS
Tetrachloroethene	C-B2	not specified	0.052	ECAO
Trichloroethene	C-B2	not specified	0.011	ECAO
Barium	NA	NA	NA	IRIS
Manganese	NA	NA	NA	IRIS

<sup>a</sup> IRIS, Integrated Risk Information System; HEAST, Health Effects Assessment Summary Tables; ECAO, Environmental Criterion Assessment Office of EPA.

<sup>b</sup> The weight-of-evidence classification and oral slope factor for Aroclor 1260 have been extrapolated from the weight-of-evidence classification and oral slope factor for PCBs.

NA Not available.

**TABLE 2-9. EPA WEIGHT-OF-EVIDENCE CLASSIFICATION SYSTEM FOR CARCINOGENICITY**

GROUP	DESCRIPTION
A	Human carcinogen.
B1 or B2	Probable human carcinogen.
	B1 indicates that limited human data are available.
	B2 indicates sufficient evidence in animals and inadequate or no evidence in humans.
C	Possible human carcinogen.
D	Not classifiable as to human carcinogenicity.
E	Evidence of noncarcinogenicity for humans.

classes A, B1, and B2. Quantitative estimation of SFs for the chemicals in class C is done on a case-by-case basis.

Generally, the SF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The SF is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. SFs should always be accompanied by the weight-of-evidence classification to indicate the strength of the evidence that the agent is a human carcinogen.

**2.3.2.4 Identifying the Appropriate Data Set.** In deriving SFs, the available information about a chemical is evaluated, and an appropriate data set is selected; human data of high quality are preferable to animal data. If animal data are used, the species that responds most similarly to humans (with respect to factors such as metabolism, physiology, and pharmacokinetics) is preferred. When no clear choice is possible, the most sensitive species is given the greatest emphasis. Occasionally, in situations where no single study is judged most appropriate yet several studies collectively support the estimate, the geometric mean of estimates from all studies may be adopted as the SF. This practice ensures the inclusion of all relevant data.

**2.3.2.5 Extrapolating to Lower Doses.** Because risk at low exposure levels is difficult to measure directly either by animal experiments or by epidemiologic studies, the development of an SF generally entails applying a model to the available data set and using the model to extrapolate from the relatively high doses administered to experimental animals (or the exposures noted in epidemiologic studies) to the lower exposure levels expected for human contact in the environment.

A number of mathematical models and procedures have been developed to extrapolate from carcinogenic responses observed at high doses to responses expected at low doses. Different extrapolation methods may provide a reasonable fit to the observed data but may lead to large differences in the projected risk at low dose.

In general, after the data are fit to the appropriate model, the upper 95th percent confidence limit of the slope of the resulting dose-response curve is calculated. This value is known as the SF and represents an upper 95th percent confidence limit of the probability of a response per unit intake of a chemical over a lifetime (i.e., there is only a five percent chance that the probability of a response could be greater than the estimated value on the basis of the experimental data and model used). In some cases, SFs based on human dose-response data are based on the "best" estimate instead of the upper 95th percent confidence limits. Because the dose-response curve generally is linear only in the low-dose region, the SF estimate only holds true for low doses. Information concerning the limitations on use of SFs can be found in IRIS.

**2.3.2.6 Summary of Dose-Response Parameters.** Toxicity values for carcinogenic effects can be expressed in several ways. The SF generally is considered to be the upper 95th percent confidence limit of the slope of the dose-response curve and is expressed as (mg/kg-day)<sup>-1</sup>. Thus:

$$\begin{aligned}\text{Slope factor} &= \text{risk per unit dose} \\ &= \text{risk per mg/kg-day}\end{aligned}$$

Where data permit, SFs listed in IRIS are based on absorbed doses, although many of them have been based on administered doses.

### **2.3.3 Summaries of the Toxicity of the Chemicals of Concern**

Tables 2-7 and 2-8 present chronic cancer and noncancer health effects criteria (oral slope factors and RfDs respectively) for the COCs. The toxicological properties of the COCs and the toxicological basis of the health effects criteria listed in Tables 2-7 and 2-8 are discussed in Appendix B.

## **2.4 RISK CHARACTERIZATION**

In the risk characterization, the toxicity and exposure assessments are summarized and integrated into quantitative and qualitative expressions of risk. To characterize potential noncancerous effects, comparisons are made between projected intakes (ADD) of substances and toxicity values (e.g., the RfD). To characterize potential carcinogenic effects, probabilities that an individual will develop cancer over a lifetime of exposure are estimated from projected intakes (LADD) and chemical-specific dose-response information (e.g., the SF). Major assumptions, scientific judgements and, to the extent possible, estimates of the uncertainties embodied in the assessment are also presented. In this section methods of quantifying risks are discussed and applied to individual sites at the Cape Lisburne installation.

## 2.4.1 Quantifying Risks

This section describes the steps for quantifying risk or hazard indices for both carcinogenic and noncancerous effects to be applied to each exposure pathway analyzed. The first two subsections cover procedures for individual substances and are followed by a subsection on procedures for quantifying risks associated with simultaneous exposures to several substances.

**2.4.1.1 Risks from Individual Substances - Carcinogenic Effects.** For carcinogens, risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen (i.e., incremental or excess individual lifetime cancer risk). The guidelines provided in this section are consistent with EPA (1986b). For some carcinogens, there may be sufficient information on the mechanism of action that a modification of the approach outlined below is warranted. Alternative approaches may be considered in consultation with ECAO on a case-by-case basis.

The SF converts estimated daily intakes averaged over a lifetime of exposure directly to incremental risk of an individual developing cancer. Because relatively low intakes (compared to those experienced by test animals) are most likely from environmental exposures, it generally can be assumed that the dose-response relationship will be linear in the low-dose portion of the multistage model dose-response curve. Under this assumption, the SF is a constant, and risk will be directly related to intake. Thus, the linear form of the carcinogenic risk equation is usually applicable for estimating cancer risks. This linear low-dose equation is described below.

### LINEAR LOW-DOSE CANCER RISK EQUATION

$$\text{Risk} = \text{LADD} \times \text{SF}$$

where:

Risk = a unitless probability (e.g.,  $2 \times 10^{-5}$ ) of an individual developing cancer;

LADD = lifetime average daily dose averaged over 70 years (mg/kg-day); and

SF = slope factor, expressed in (mg/kg-day)<sup>-1</sup>.

Because the SF is often an upper 95th percentile confidence limit of the probability of response based on experimental animal data used in the multistage model, the carcinogenic risk estimate generally will be an upper-bound estimate. This means that the "true risk" will probably not exceed the risk estimate derived through use of this model and is likely to be lower than predicted.

**2.4.1.2 Noncancer Hazards from Individual Substances - Noncancerous Effects.** The measure used to describe the potential for noncancerous toxicity in an individual is not expressed as the probability of an individual suffering an adverse effect. EPA does not at the present time use a probabilistic approach to estimate the potential for noncancerous health effects. Instead, the potential for noncancerous effects is evaluated by comparing an exposure level over a specified time period (e.g., some fraction of a lifetime) with an RfD derived for a similar exposure period. This ratio of exposure to toxicity is called an HQ.

The noncancer HQ assumes there is a level of exposure (i.e., the RfD) below which it is unlikely even for sensitive populations to experience adverse health effects. If the exposure level (ADD) exceeds this threshold (i.e., if ADD/RfD exceeds unity), there may be concern for potential noncancer effects. As a rule, the greater the value of ADD/RfD above unity, the greater the level of concern. Ratios of ADD/RfD should not be interpreted as statistical probabilities; a ratio of 0.001 does not mean that there is a one in one thousand chance of the effect occurring. Further, it is important to emphasize that the level of concern does not increase linearly as the RfD is approached or exceeded because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects. Thus, the slopes of the dose-response curve in excess of the RfD can range widely depending on the substance.

### NONCANCER HAZARD QUOTIENT

$$\text{Noncancer Hazard Quotient} = \text{ADD/RfD}$$

where:

ADD = average daily dose (or intake);

RfD = reference dose

ADD and RfD are expressed in the same units and represent the same exposure period (e.g., chronic, subchronic, or short-term).

**2.4.1.3 Aggregate Risks for Multiple Substances.** Estimating risk or hazard potential by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several substances. To assess the overall potential for cancer and noncancer effects posed by multiple chemicals, EPA has developed *Guidelines for the Health Risk Assessment of Chemical Mixtures* (EPA 1986b). These guidelines can be applied to the case of simultaneous exposures to several chemicals from a variety of sources by more than one exposure pathway. Information on specific mixtures is rarely available. Even if such data exist, they are often difficult to use. Monitoring for "mixtures" or modeling the movement of mixtures across space and time presents significant technical problems given the likelihood that individual components will behave differently in the environment (i.e., fate and transport).

Although the calculation procedures differ for carcinogenic and noncarcinogenic effects, both sets of procedures assume dose additivity in the absence of information on specific mixtures.

**Carcinogenic effects.** The cancer risk equation described below is used to estimate the incremental individual lifetime cancer risk for simultaneous exposure to several carcinogens based on EPA's risk assessment guidelines. This equation represents an approximation of the precise equation for combining risks that accounts for the joint probabilities of the same individual developing cancer as a consequence of exposure to two or more carcinogens. The difference between the precise equation and the approximation described in the equation below is negligible for total cancer risks less than 0.1. Thus, the simple additive equation is appropriate for most risk assessments.



## CANCER RISK EQUATION FOR MULTIPLE SUBSTANCES

$$\text{Risk}_T = \Sigma \text{Risk}_i$$

where:

$\text{Risk}_T$  = the total cancer risk, expressed as a unitless probability; and

$\text{Risk}_i$  = the risk estimate for the  $i^{\text{th}}$  substance.

The risk summation techniques described in the cancer risk equation above assume that intakes of individual substances are small. They also assume independence of action by the compounds involved (i.e., that there are no synergistic or antagonistic chemical interactions and that all chemicals produce the same effect, i.e., cancer). If these assumptions are incorrect, over- or under-estimation of the actual multiple-substance risk could result.

A separate total cancer risk for each exposure pathway is calculated by summing the substance-specific cancer risks. Resulting cancer risk estimates should be expressed using one significant figure only.

There are several limitations to this approach. First, because each SF is an upper 95th percentile estimate of potency and upper 95th percentiles of probability distributions are not strictly additive, the total cancer risk estimate might become artificially more conservative as risks from a number of different carcinogens are summed. If one or two carcinogens drive the risk, however, this problem is not of concern. Second, it often will be the case that substances with different weights of evidence for human carcinogenicity are included. The cancer risk equation for multiple substances sums all carcinogens equally, giving as much weight to class B or C as to class A carcinogens. In addition, SFs derived from animal data will be given the same weight as SFs derived from human data. Finally, the action of two different carcinogens might not be independent.

**Noncancerous effects.** To assess the overall potential for noncancerous effects posed by more than one chemical, a hazard index approach has been developed based on EPA's *Guidelines for Health Risk Assessment of Chemical Mixtures* (EPA 1986b). This approach assumes that simultaneous subthreshold exposures to several chemicals could result in an adverse health effect. It also assumes that the magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures. The hazard index is equal to the sum of the HQs. When the hazard index exceeds unity, there may be concern for potential health effects. Any single chemical with an exposure level greater than the toxicity value will cause the hazard index to exceed unity, and for multiple chemical exposures, the hazard index can exceed unity even if no single chemical exposure exceeds its RfD. The equation used to determine noncancer hazard index is as follows:

### NONCANCER HAZARD INDEX

$$\text{Hazard Index} = \text{ADD}_1/\text{RfD}_1 + \text{ADD}_2/\text{RfD}_2 + \dots + \text{ADD}_i/\text{RfD}_i$$

where:

$\text{ADD}_i$  = average daily dose (or intake) for the  $i^{\text{th}}$  toxicant;

$\text{RfD}_i$  = reference dose for the  $i^{\text{th}}$  toxicant; and

ADD and RfD are expressed in the same units and represent the same exposure period (i.e., chronic, subchronic, or shorter-term).

Where appropriate, a separate chronic hazard index can be calculated from the ratios of the chronic daily intake (CDI) to the chronic RfD for individual chemicals as described below.

### CHRONIC NONCANCER HAZARD INDEX

$$\text{Chronic Hazard Index} = \text{LADD}_1/\text{RfD}_1 + \text{LADD}_2/\text{RfD}_2 + \dots + \text{LADD}_i/\text{RfD}_i$$

where:

$\text{LADD}_i$  = lifetime average daily dose for the  $i^{\text{th}}$  toxicant in mg/kg-day, and

$\text{RfD}_i$  = chronic reference dose for the  $i^{\text{th}}$  toxicant in mg/kg-day.

There are several limitations to this approach. As mentioned earlier, the level of concern does not increase linearly as the RfD is approached or exceeded because the RfDs do not have equal accuracy or precision and are not based on the same severity of effect. Moreover, HQs are combined for substances with RfDs based on critical effects of varying toxicological significance. It will often be the case that RfDs of varying levels of confidence that include different uncertainty adjustments and modifying factors will be combined (e.g., extrapolation from animals to humans, from lowest-observed adverse effect levels (LOAELs) to no observed adverse effect levels (NOAELs), from one exposure duration to another).

Another limitation with the hazard index approach is that the assumption of dose additivity is most properly applied to compounds that induce the same effect by the same mechanism of action. Consequently, application of the hazard index equation to a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism could overestimate the potential for effects, although such an approach is appropriate at a screening level. This possibility is generally not of concern if only one or two substances are responsible for driving the hazard index above unity. If the hazard index is greater than unity as a consequence of summing several HQs of similar value, it is appropriate to segregate the compounds by effect and by mechanism of action and derive separate hazard indices for each group.

#### 2.4.2 Site-Specific Risk Characterization

**Soil and Sediment Exposures.** The quantification of noncancer hazard and excess lifetime cancer risk associated with the soil ingestion pathway at Cape Lisburne was based on analytical

data from soil and sediment samples collected within the interval from ground surface to permafrost. No attempt was made to segregate surface soil samples from subsurface samples in the risk characterization.

The noncancer hazard and the excess lifetime cancer risk associated with the ingestion of soil or sediment containing COCs has been estimated for an hypothetical native northerner based on six years of exposure as a child and 49 years of exposure as an adult. For the radar installation worker, cancer risk has been estimated based on ten years of exposure averaged over a default lifetime of 70 years. Noncancer hazard for the radar installation worker was based on a 10 year exposure.

**Surface Water Exposures.** The noncancer hazard and the excess lifetime cancer risk associated with the ingestion of surface water containing COCs has been estimated based on a native northern adult and a radar installation worker. A native northern child receptor was not considered because, unlike exposure to soil, which is expected to be greater in a child than in an adult, the ratio of drinking water ingestion rate to body weight is assumed to be relatively constant from childhood to adulthood. A greater number of years is spent as an adult, so estimating hazard or risk for water ingestion based on an adult is a more conservative approach. The exposure duration estimate for the radar installation worker was 10 years and for the native northern adult was 55 years. Exposures were averaged over 10 years for a radar installation worker exposure to noncarcinogens, and 55 years for native northern adult exposure to noncarcinogens. Exposures were averaged over 70 years for both receptor groups to characterize the risk associated with exposure to carcinogens in surface water.

Ingestion of surface water at the Cape Lisburne installation is not considered to be a complete pathway under a current use scenario; water for domestic use is obtained from the Water Gallery (AOC3) which is upgradient of areas that may have been impacted by the installation. Sampling at the Water Gallery has confirmed the absence of any impact on the installation water supply.

Under a future use scenario, however, it is possible that the buildings could be used for residences or additional residential structures could be erected at the installation. The future residents could be either radar installation workers or native northerners. Therefore, because sources of water may change in the future, potential ingestion of surface water at the installation will be evaluated for the radar installation worker and native northern adult under a future use exposure scenario only.

Table 2-10 contains a site-by-site summary of the COCs in each medium, and the noncancer hazard and excess lifetime cancer risk associated with exposure to the COCs in the soils, sediments, and surface water. COCs without toxicity data (RBSLs or ARARs) are not included on Table 2-10, but are discussed in Section 2.1.5. Appendix A contains the spreadsheets used to calculate the noncancer hazard and excess lifetime cancer risk estimates presented in Table 2-10.

**Risk Characterization of Petroleum Hydrocarbons.** Petroleum hydrocarbons represent a potentially important source of contamination at the Cape Lisburne installation. The laboratory analysis of soil, sediment, and surface water samples revealed the presence of DRPH and RRPH.

TABLE 2-10. SUMMARY OF NONCANCER HAZARD AND EXCESS LIFETIME CANCER RISK FOR CAPE LISBURNE

SITE	MEDIUM	NONCANCER CHEMICALS OF CONCERN <sup>a</sup>	NONCANCER HAZARD <sup>c</sup>			CARCINOGENIC CHEMICALS OF CONCERN	EXCESS LIFETIME CANCER RISK <sup>d</sup>		
			RADAR INSTALLATION WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD		RADAR INSTALLATION WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD
Landfill and Waste Accumulation Area (LF01)	Soil or Sediment	DRPH RRPH Aroclor 1260 Carbon tetrachloride	3	— <sup>e</sup>	62	Carbon tetrachloride Aroclor 1260 Trichloroethene	6 x 10 <sup>-5</sup>	— <sup>e</sup>	1 x 10 <sup>-3</sup>
	Surface Water	Carbon tetrachloride Tetrachloroethene Chloroform Barium Manganese	7	7 <sup>f</sup>	—	Benzene Tetrachloroethene Carbon tetrachloride Trichloroethene Chloroform	2 x 10 <sup>-5</sup>	1 x 10 <sup>-4</sup>	—
White Alice Site (SS03)	Soil or Sediment	Aroclor 1260	18	—	381	Aroclor 1260	4 x 10 <sup>-4</sup>	—	9 x 10 <sup>-3</sup>
	Surface Water	NONE <sup>b</sup>	—	—	—	NONE	—	—	—
Spill/Leak #3 (ST07)	Soil or Sediment	DRPH GRPH	0.05	—	1.0	GRPH Benzene	2 x 10 <sup>-9</sup>	—	5 x 10 <sup>-8</sup>
	Surface Water	NONE	—	—	—	NONE	—	—	—
Upper Camp Transformer Building (SS08)	Soil or Sediment	DRPH RRPH Aroclor 1254 Aroclor 1260	1.0	—	21	Aroclor 1254 Aroclor 1260	2 x 10 <sup>-5</sup>	—	4 x 10 <sup>-4</sup>
	Surface Water	NONE	—	—	—	NONE	—	—	—
Lower Camp Transformer Buildings (SS09)	Soil or Sediment	DRPH RRPH Aroclor 1254 Aroclor 1260	22	—	445	Aroclor 1254 Aroclor 1260	5 x 10 <sup>-4</sup>	—	1 x 10 <sup>-2</sup>
	Surface Water	NONE	—	—	—	NONE	—	—	—

Pathway not evaluated.

All COCs are listed together regardless of whether they contribute to the hazard index, cancer risk, or both.

None, no COCs selected.

Hazard index, noncancer hazard index. The hazard index is the sum of the HQs for all of the COCs associated with a given medium, pathway, and receptor group.

Cancer risk, excess lifetime cancer risk. The cancer risk is the sum of the excess lifetime cancer risks for all of the carcinogenic COCs associated with a given medium, pathway, and receptor group.

**TABLE 2-10. SUMMARY OF NONCANCER HAZARD AND EXCESS LIFETIME CANCER RISK FOR CAPE LISBURNE (CONTINUED)**

Children are assumed to have a soil ingestion rate greater than that for adults. Therefore, under a residential scenario, the estimates of noncancer hazard and cancer risk associated with soil ingestion are estimated for a combined adult and child receptor only. This estimate is considered a conservative upper bound on the true hazard or risk. Drinking water ingestion, unlike soil ingestion, is evaluated for an adult receptor but not a child receptor because adults are assumed to have a longer exposure duration at a greater water ingestion rate. Therefore, the hazard or risk estimated will represent an upper bound, conservative estimate. For soil ingestion, the child soil ingestion rate is assumed to exceed that for adults. Therefore, a combination of the adult and child receptor groups is used to evaluate soil ingestion risk and hazard.

To characterize the risk associated with exposure to these compounds, we applied the provisional RfDs and the SF developed by EPA for petroleum hydrocarbons (EPA 1992b). These provisional RfDs provide the best available tool for characterizing the risk associated with exposure to the petroleum hydrocarbons. The RfD for JP-4 presented in EPA (1992b) was used to represent DRPH and RRPH.

The noncancer hazard associated with exposure to DRPH and RRPH was, therefore, estimated by dividing the compound- and site-specific ADD by the appropriate provisional RfD (EPA 1992b).

Although the provisional RfDs represent the best available numerical estimate of toxicity, there is a significant amount of uncertainty associated with their use at the Cape Lisburne installation. The RfDs are based on studies in mice and rats by the inhalation route of exposure; whereas, in this risk assessment, exposure of humans by the ingestion route is being evaluated.

Furthermore, in the absence of a more thorough study to compare the DRPH and RRPH to known petroleum refinery streams, it is not clear how well the provisional values represent the toxicity of diesel and gasoline in humans.

**Risk Characterization of Chemicals Detected.** Chemicals detected above background levels without RBSLs or ARARs are evaluated in Section 2.1.5 (page 2-14). Based on the information in that section, and the relatively low levels detected at the sites, these chemicals are not expected to pose a health risk. Risk characterization of chemicals detected that exceed RBSLs, ARARs, or both are discussed on a site-by-site basis below.

#### **2.4.2.1 Landfill and Waste Accumulation Area (LF01).**

**Soils and Sediments.** The noncancer hazard associated with the ingestion of soil at Landfill and Waste Accumulation Area by a hypothetical native northern adult/child is 62, and by a radar installation worker is 3, based on the maximum concentrations of the COCs (Tables 2-10 and A-1). The presence of Aroclor 1260 accounts for more than 99 percent of the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at the site by a hypothetical native northern adult/child is  $1 \times 10^{-3}$ , and by a radar installation worker is  $6 \times 10^{-5}$ , based on the maximum concentrations of the COCs (Tables 2-10 and A-2). The presence of Aroclor 1260 accounts for more than 99 percent of the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

**Surface Water.** The noncancer hazard associated with the ingestion of surface water at the site by a hypothetical native northern adult and by a radar installation worker is 7, based on the maximum concentrations of the COCs (Tables 2-10 and A-3). Carbon tetrachloride, tetrachloroethene, chloroform, barium, and manganese account for the quantifiable noncancer hazard for these receptor/pathway combinations. Manganese alone accounts for more than 99 percent of the noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of surface water at the site by a native northern adult is  $1 \times 10^{-4}$ , and by a radar installation worker is  $2 \times 10^{-5}$ , based on the maximum concentrations of the COCs (Tables 2-10 and A-4). The presence of benzene, tetrachloroethene, carbon tetrachloride, trichloroethene, and chloroform account for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

#### **2.4.2.2 White Alice Site (SS03).**

**Soils and Sediments.** The noncancer hazard associated with the ingestion of soil at the White Alice Site (SS03) by a hypothetical native northern adult/child is 381, and by a radar installation worker is 18, based on the maximum concentration of the COC (Tables 2-10 and A5). The presence of Aroclor 1260 accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at the site by a hypothetical native northern adult/child is  $9 \times 10^{-3}$ , and by a radar installation worker is  $4 \times 10^{-4}$ , based on the maximum concentration of the COC (Tables 2-10 and A-6). The presence of Aroclor 1260 accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

**Surface Water.** No surface water bodies were identified at the site, therefore no evaluation of noncancer hazard or excess lifetime cancer risk associated with ingestion of surface water was conducted.

#### **2.4.2.3 Spill/Leak #3 (ST07).**

**Soils and Sediments.** The noncancer hazard associated with the ingestion of soil at the Spill/Leak #7 (ST07) by a hypothetical native northern adult/child is 1.0, and by a radar installation worker is 0.05, based on the maximum concentrations of the COCs (Tables 2-10 and A-7). The presence of DPRH and GRPH accounts for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at the site by a hypothetical native northern adult/child is  $5 \times 10^{-8}$ , and by a radar installation worker is  $2 \times 10^{-9}$ , based on the maximum concentrations of the COCs (Tables 2-10 and A-8). The presence of GRPH and benzene accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

**Surface Water.** No COCs were identified for the surface water at the site (Table 2-1). This does not indicate that exposure to chemicals in the surface water at the site is without health risk; however, the concentrations measured were lower than the concentrations considered acceptable under Region 10 guidance (EPA 1991a) or federal ARARs.

#### **2.4.2.4 Upper Camp Transformer Building (SS08).**

**Soils and Sediments.** The noncancer hazard associated with the ingestion of soil at the Upper Camp Transformer Building (SS08) by a hypothetical native northern adult/child is 21, and by a radar installation worker is 1, based on the maximum concentrations of the COCs (Tables 2-10 and A-10). The presence of DPRH, RRPB, Aroclor 1254, and Aroclor 1260 accounts for the quantifiable noncancer hazard for these receptor/pathway combinations. Aroclors 1254 and 1260 account for more than 99 percent of the noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at the site by a hypothetical native northern adult/child is  $4 \times 10^{-4}$ , and by a radar installation worker is  $2 \times 10^{-5}$ , based on the maximum concentrations of the COCs (Tables 2-10 and A-11). The presence of Aroclors 1254 and 1260 accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

**Surface Water.** No surface water bodies were identified at the site; therefore, no evaluation of noncancer hazard or excess lifetime cancer risk associated with ingestion of surface water was conducted.

#### **2.4.2.5 Lower Camp Transformer Buildings (SS09).**

**Soils and Sediments.** The noncancer hazard associated with the ingestion of soil at the Lower Camp Transformer Buildings (SS09) by a hypothetical native northern adult/child is 445, and by a radar installation worker is 22, based on the maximum concentrations of the COCs (Tables 2-10 and A-12). The presence of DPRH, RRPB, Aroclor 1254, and Aroclor 1260 accounts for the quantifiable noncancer hazard for these receptor/pathway combinations. The Aroclors alone account for more than 99 percent of the noncancer hazard.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at the site by a hypothetical native northern adult/child is  $1 \times 10^{-2}$ , and by a radar installation worker is  $5 \times 10^{-4}$ , based on the maximum concentrations of the COCs (Tables 2-10 and A-13). The presence of Aroclor 1254 and Aroclor 1260 accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

**Surface Water.** No surface water bodies were identified at the site; therefore, no evaluation of noncancer hazard or excess lifetime cancer risk associated with ingestion of surface water was conducted.

### **2.5 RISK CHARACTERIZATION UNCERTAINTY**

Several sources of uncertainty affect the estimates of excess lifetime cancer risk and noncancer hazard as presented in this risk assessment. The sources are generally associated with:

- Sampling and analysis of soil, sediment, and surface water;



- Assigning the source of contamination;
- Exposure assumptions, including estimates of exposure point concentrations;
- Evaluation of the toxicity of the COCs; and
- Methods and assumptions used to characterize the cancer risk and noncancer hazard.

Uncertainties associated with sampling and analysis include the inherent variability (standard error) in the analysis, representativeness of the samples, sampling errors, and heterogeneity of the sample matrix. The quality assurance/quality control program used in conducting the sampling and analysis serves to reduce errors, but it can not eliminate all errors associated with sampling and analysis. There is some uncertainty in the selection of COCs with respect to sample quantitation limits for a given chemical. In some cases a chemical may have had detected values below the COC screening criteria as well as samples with quantitation limits greater than the screening criteria. In these cases it should be understood that only the samples with adequate quantitation limits are applicable to the screening process. Thus, the number of samples used to screen a chemical would be less than the total number of analyses for that chemical.

Simplifying assumptions were made about the environmental fate and transport of site contamination, specifically, no contaminant loss or transformation has or will occur. Thus, the data chosen to represent exposure point concentrations in the sample-by-sample risk calculations are an additional source of potential error.

The depth at which a soil sample was collected was not considered in the risk characterization, so exposure to subsurface contamination was considered to be equally likely as exposure to surface contamination. This approach would tend to overestimate the true risk.

The estimation of exposure requires many assumptions to describe potential exposure situations. There are uncertainties regarding the likelihood of exposure, frequency of contact with contaminated media, the concentration of contaminants at exposure points, and the time period of exposure. These tend to simplify and approximate actual site conditions. In general, these assumptions are intended to be conservative and yield an overestimate of the true risk or hazard.

The toxicological database is also a source of uncertainty. The EPA has outlined some of the sources of uncertainty in the data base (EPA 1986a,b, 1989a). These sources include extrapolation between exposure routes from high to low doses and from animals to humans; species, gender, age, and strain differences in uptake, metabolism, organ distribution, and target site susceptibility; and human population variability with respect to diet, environment, activity patterns, and cultural factors. The toxicity factors from IRIS and HEAST, which are used to estimate the toxicity of the COCs, are developed using a highly conservative methodology and probably tend to overestimate the potential hazards to humans.

Use of the provisional RfDs and SFs for DRPH, GRPH, and RRPB are an additional source of uncertainty in the toxicity assessment and risk characterization. Although the provisional RfDs represent the best available numerical estimate of toxicity, there is a significant amount of uncertainty associated with their use at the Cape Lisburne installation. The RfDs and SFs are based on studies in mice and rats by the inhalation route of exposure; whereas, in this risk assessment, exposure of humans by the ingestion route only is being evaluated. Furthermore, in the absence of more thorough studies to compare to toxicity of DRPH, GRPH, and RRPB to the toxicity of known refinery streams, it is not clear how well the provisional values represent the toxicity of diesel, gasoline, and residual oils in humans.

In the risk characterization, the assumption was made that the total risk of developing cancer from exposure to site contaminants is the sum of the risk attributed to each individual contaminant. Likewise, the potential for the development of noncancer adverse effects is the sum of the HQs estimated for exposure to each individual contaminant. This approach does not account for the possibility that chemicals act synergistically or antagonistically but probably results in an overestimate of the true risk.

In addition to the more general sources of uncertainty associated with risk assessment methodology, there are site-specific sources of uncertainty. Primarily, these sources are associated with the lifestyle of the native northerners, the time spent on the sites that were investigated during the RI, and specific exposure assumptions (soil ingestion rate, exposure frequency, and exposure duration).

Because no roads connect Point Hope to Cape Lisburne, inhabitants of Point Hope are not likely to use the installation as an access route for recreation or subsistence hunting. No studies have been conducted to measure the time these potential receptors spend on contaminated sites at the installation; however, the time is probably limited to occasional visits to the installation via boat during the summer months and snowmobile in winter. Some of the sites with levels of contamination that exceed regulatory benchmarks are not likely to be accessed by this group. Therefore, the assumptions made regarding exposure frequency probably result in an overestimate of the true noncancer hazard and cancer risk.

The risk characterization also included consideration of use of surface water as a drinking water supply. The Water Gallery (AOC3) is a surface water and shallow ground water source that currently supplies the installation. As indicated in the analytical results from samples collected at the Water Gallery, this is a clean supply with no contamination from the installation. No other surface water body is used as a domestic supply. Therefore, a significant source of uncertainty in the risk assessment is based on the assumption that other surface water sources might be used as a domestic supply in the future. This approach will result in an overestimate of cancer risk, noncancer hazard, or both.

Similarly, no studies have been conducted to measure the soil ingestion rate of potential receptors on the contaminated sites. Soil ingestion by potential future inhabitants at Cape Lisburne (assuming a potential residential scenario) may be greater than the default rate of 100 mg/day for adults and 200 mg/day for children. Given the rugged, partially subsistence lifestyle of this group, it is possible that they incidentally ingest soil at a higher rate than receptors

of a similar age in the continental United States. The estimate of soil ingestion rate used in this risk assessment may over- or underestimate the true rate.

The maximum exposure duration assumed for native northerners, 55 years, is probably fairly accurate. The reasonable maximum exposure estimate for inhabitants of the continental United States is 30 years; however, native northerners are more likely to remain in their villages for a longer period. Although, the exposure duration of 55 years is an estimate, it is not expected to significantly over- or underestimate hazard or risk.

## 2.6 RISK ASSESSMENT SUMMARY AND CONCLUSIONS

The potential human health risks associated with exposure to contaminated media (soil, sediment, or surface water) at five sites at the Cape Lisburne radar installation were evaluated in this risk assessment. The risk assessment was developed using a three step process:

- 1) The maximum concentrations of the chemicals detected in each medium (soil, sediment, or surface water) were compared to background concentrations, RBSLs, and ARARs. Chemicals present at concentrations that exceeded their background concentration and either an RBSL or an ARAR were retained as COCs for the risk assessment.
- 2) In the risk characterization, the noncancer HQ, the excess lifetime cancer risk, or both were calculated based on the maximum concentration of each COC and its associated toxicity value developed by EPA.
- 3) The HQs for each COC at a given site were then summed, and the sum (called a hazard index) was compared to the regulatory benchmark for noncancer hazard: a hazard index of 1. Sites where the hazard index exceeded 1 are considered to warrant either remediation or further discussion. Sites where the hazard index was less than 1 are considered to warrant no further action (EPA 1991c).

The cancer risks for each carcinogenic COC at a given site were also summed, and the sum (the total cancer risk) was compared to the regulatory benchmark for cancer risks: an excess lifetime cancer risk of  $1 \times 10^{-6}$  (one in one million). Sites where the total cancer risk exceeded  $1 \times 10^{-6}$  are considered to warrant either remediation or further discussion (EPA 1991c). Sites where the total cancer risk was less than  $1 \times 10^{-6}$  are considered to warrant no further action.

Table 2-11 contains a summary of the noncancer hazard and excess lifetime cancer risk for each site and medium at the Cape Lisburne installation that exceeds a regulatory benchmark of 1.0 for noncancer hazard index or  $1 \times 10^{-6}$  for excess lifetime cancer risk.

**No Further Action.** Based on the quantification of noncancer hazard and excess lifetime cancer risk, only one of the five sites investigated at Cape Lisburne warrants no further action: Spill/Leak #3 (ST07). The cumulative noncancer hazard is less than 1.0 and the excess lifetime cancer risk

**TABLE 2-11. SUMMARY OF SITES WITH CONTAMINATION THAT EXCEEDS REGULATORY BENCHMARKS**  
**[Noncancer Hazard Index > 1.0, Excess Lifetime Cancer Risk >  $1 \times 10^{-6}$ ]**

SITE	MEDIUM	NONCANCER CHEMICALS OF CONCERN <sup>a</sup>	NONCANCER HAZARD			CARCINOGENIC CHEMICALS OF CONCERN <sup>a</sup>	EXCESS LIFETIME CANCER RISK		
			RADAR INSTALLATION WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD		RADAR INSTALLATION WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD
Landfill and Waste Accumulation Area (LF01)	Soil or Sediment	DRPH RRPH Aroclor 1260 Carbon tetrachloride	3	—	62	Carbon tetrachloride Aroclor 1260 Trichloroethene	$6 \times 10^{-5}$		$1 \times 10^{-3}$
	Surface Water	Carbon tetrachloride Tetrachloroethene Chloroform Barium Manganese	7	7	—	Benzene Tetrachloroethene Carbon tetrachloride Trichloroethene Chloroform	$2 \times 10^{-5}$	$1 \times 10^{-4}$	—
White Alice Site (SS03)	Soil or Sediment	Aroclor 1260	18	—	381	Aroclor 1260	$4 \times 10^{-4}$	—	$9 \times 10^{-3}$
Spill/Leak #3 (ST07)	Soil or Sediment	DRPH GRPH	—	—	—	GRPH Benzene	—	—	—
Upper Camp Transformer Building (SS08)	Soil or Sediment	DRPH RRPH Aroclor 1254 Aroclor 1260	—	—	21	Aroclor 1254 Aroclor 1260	$2 \times 10^{-5}$	—	$4 \times 10^{-4}$
Lower Camp Transformer Buildings (SS09)	Soil or Sediment	DRPH RRPH Aroclor 1254 Aroclor 1260	22	—	445	Aroclor 1254 Aroclor 1260	$5 \times 10^{-4}$	—	$1 \times 10^{-2}$

<sup>a</sup> All COCs are listed regardless of whether they contribute to the hazard index, or cancer risk.

is less than  $1 \times 10^{-6}$  for the radar installation worker and the native northern adult/child receptor groups. Highly conservative (residential) exposure assumptions were applied to the estimation of noncancer hazard and cancer risk, and the analytical samples, on which the estimates of hazard and risk are based, were collected from areas expected to be contaminated. Therefore, it is not likely that the Spill/Leak #3 (ST07) site poses a threat to human health.

**Sites that Warrant Further Discussion.** Based on the noncancer hazard or excess lifetime cancer risk associated with the ingestion of soil/sediment and/or surface water, sites that warrant further discussion are:

- Landfill and Waste Accumulation Area (LF01) - soil/sediment and surface water
- White Alice Site (SS03) - soil/sediment only
- Upper Camp Transformer Building (SS08) - soil/sediment only
- Lower Camp Transformer Buildings (SS09) - soil/sediment only

A site is considered to warrant further discussion if the cumulative noncancer hazard or excess lifetime cancer risk associated with exposure to the soil/sediment or surface water exceeds the regulatory benchmarks of 1.0 for noncancer hazard and  $1 \times 10^{-6}$  for excess lifetime cancer risk.

The magnitude of the cumulative noncancer hazard and excess lifetime cancer risk associated with the soil or sediment at each site that warrants further discussion is largely due to high concentrations of Aroclors 1254 and 1260. To determine, in part, whether remedial action may be necessary, the likelihood of exposure must be considered. The most significant noncancer hazards and cancer risks are associated with samples collected inside the Lower Camp Transformer Buildings. Exposure to the Aroclors at this site is not likely to occur under the worker or residential assumptions applied in this risk assessment: the buildings have been deactivated and the transformers and other equipment removed. Similarly, exposure to Aroclor-contaminated soil at the Upper Transformer Building and the White Alice Site is not likely to occur under worker or residential assumptions because these building have been deactivated and the transformers and other equipment removed.

Under a future use scenario, it is possible that the transformer buildings and the White Alice structure could be used for occupational purposes. Therefore remedial action at these sites may be warranted.

The presence of Aroclor 1260 in soils and sediments at the Landfill and Waste Accumulation Area yields the most significant portion of the cumulative noncancer hazard and excess lifetime cancer risk for this site. Based on the magnitude of the noncancer hazard and cancer risk associated with this site, remedial action work may be warranted.

The cumulative noncancer hazard and excess lifetime cancer risk associated with the surface water at the Landfill and Waste Accumulation Area exceed the regulatory benchmarks. Manganese and carbon tetrachloride contribute more than 99 percent of the noncancer hazard, and carbon tetrachloride alone contributes more than 99 percent of the excess lifetime cancer risk. As noted in the exposure assessment section of this risk assessment report, surface water impacted by site contaminants is not used for domestic or occupational purposes and is not

likely to be used in the future. The Water Gallery, in an area not impacted by activities at the installation, provides a potable water supply. Thus, the surface water at the Landfill and Waste Accumulation Area does not necessarily warrant remedial action.

In conclusion based on the human health risk assessment four of the five sites at the Cape Lisburne installation may warrant remedial action. These sites include the Landfill and Waste Accumulation Area (LF01), White Alice Site (SS03), Upper Camp Transformer Building (SS08), and Lower Camp Transformer Buildings (SS09).

### 3.0 ECOLOGICAL RISK ASSESSMENT

The objective of this ERA is to estimate potential impacts to aquatic and terrestrial plants and animals at the Cape Lisburne radar installation based on sampling and analyses conducted during the RI of the sites located at the installation. The RI was completed during the summer of 1993 in conjunction with RIs at seven other radar installations.

Guidance documents used during preparation of this assessment include:

- Handbook to Support the Installation Restoration Program Statements of Work (U.S. Air Force 1991);
- Framework for Ecological Risk Assessment (EPA 1992a); and
- Ecological Risk Assessment Guidance for Superfund (EPA 1994).

The approach used to assess potential ecological impacts is conceptually similar to that for human health risks; potentially exposed populations (receptors) are identified, and then information on exposure and toxicity are combined to derive estimates of risk. The ERA focuses, however, on potential impacts to a population of organisms rather than individual organisms (except in the case of endangered species, where individuals are considered). Because ecosystems are composed of a variety of species, ERAs evaluate potential impacts to numerous species.

Ideally, ERAs should evaluate potential risks to communities and ecosystems, as well as to individual populations. Because of the large number of species and communities present in natural systems, such ecosystem-wide assessments are very complex and appropriate assessment methodologies have not yet been developed. In addition, dose-response data on community or ecosystem responses generally are lacking. Therefore, evaluations of potential impacts to communities or ecosystems are qualitative.

The degree to which potential ecological impacts can be characterized is highly dependent upon the data available to support such estimates. Such data include: information regarding contaminant release, transport and fate; characteristics of potential receptor populations; and adequate supporting toxicity data for the chemicals evaluated.

This ERA is intended to be at a screening level, rather than a full scale investigation of the state of the ecosystem. No site-specific studies of the biota were undertaken. It is based on media sampling (i.e., surface water and soil/sediment samples), and is divided into six sections:

- Section 3.1 - Selection of Site Contaminants;
- Section 3.2 - Exposure Assessment;
- Section 3.3 - Ecological Toxicity Assessment;
- Section 3.4 - Risk Characterization for Ecological Receptors;

### 3.1 SELECTION OF SITE CONTAMINANTS

A stressor in the environment is a chemical, physical, or biological action that can cause a negative impact on an ecosystem (EPA 1992a). Only chemical stressors identified as COCs are evaluated as part of this ERA. A review of the site data indicates that the chemical stressors are primarily petroleum products, solvents, polychlorinated biphenyls, and metals.

The five sites at the Cape Lisburne installation are all considered to be potentially suitable habitat for ecological receptors.

COCs are selected based on comparisons to background concentrations and action levels [Federal Ambient Water Quality Criteria (AWQC) for surface water; ADEC Water Quality Standards (18 AAC 70.020[b]) January 1995; Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota (Suter and Mabrey 1994); ADEC determination of cleanup levels for petroleum contaminated soils (ADEC 1991); EPA sediment quality criteria (as estimated by Hull and Suter 1994); and National Oceanic and Atmospheric Administration (NOAA) Sediment Effects Range (Low) (NOAA 1991)]. If no action levels were available, the maximum detected concentration of the chemical was compared to a toxicity value derived from acute or chronic exposure tests available in the literature. If the maximum concentration was above this level, the compound was considered a COC. Chemicals present onsite at concentrations in excess of background concentrations and action levels were evaluated for frequency of detection in onsite media. If a chemical was detected at a frequency of less than five percent, it was not considered representative of actual site conditions and was eliminated from evaluation in the risk assessment. Further, an attempt was made to screen out chemicals that were within the range of natural background levels. To that end, if the average concentration (exposure concentration) of a chemical was below the maximum background concentration (i.e., if the average falls within the range of background), and if the maximum detected concentration was less than twice the maximum background concentration (which is meant to approximate the 95 percent UCL of background concentrations), the chemical was not considered a COC. Note that this criterion is used to account for outliers (i.e., hotspots) that are not representative of the distribution and concentration of chemicals to which ecological receptors may be exposed. Tables 3-1 and 3-2 present the data used in the screening process for surface water and soil/sediment. Only chemicals that were detected in at least one environmental sample are presented in these summary tables.

In summary, the decisions for selecting COCs were made using the following logic:

STEP ONE: Is the chemical detected above the maximum detected background concentration?

<b>No:</b>	Not considered a COC.
<b>Yes:</b>	Continue to step two.



TABLE 3-1. SUMMARY OF CHEMICALS OF CONCERN: SURFACE WATER

CHEMICALS OF CONCERN: CAPE LISBURNE INSTALLATION SURFACE WATER						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (µg/L)	BACKGROUND (µg/L)	ACTION LEVEL (µg/L)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (µg/L)	SELECTED AS COC
ORGANICS						
Benzene	5	<1	5,300 <sup>a</sup>	2/6	2.00	NO
Toluene	9.6	<1	17,500 <sup>a</sup>	1/6	2.02	NO
Ethylbenzene	2	<1	32,000 <sup>a</sup>	2/6	1.00	NO
Xylenes (total)	4 - 5	<2	62,308 <sup>b</sup>	3/6	2.83	NO
Carbon tetrachloride	24.7 - 89	<1	35,200 <sup>a</sup>	2/6	19.28	NO
Chloroform	3.6 - 16	<1	1,240 <sup>a</sup>	2/6	3.60	NO
cis-1,2-Dichloroethene	1.35	<1	9,538 <sup>b</sup>	1/6	0.64	NO
Naphthalene	1.60	<1	620 <sup>b</sup>	1/6	0.68	NO
Tetrachloroethene	4	<1	750 <sup>b</sup>	1/6	1.08	NO
Trichloroethene	5.5 - 62	<1	21,900 <sup>a</sup>	2/6	11.58	NO
1,2,4-Trimethylbenzene	1.15	<1	--	1/6	0.61	YES
INORGANICS - based on total metals						
Aluminum	140 - 160	<100	460 <sup>b</sup>	2/4	100	NO
Barium	77 - 860	79 - 92	5,800 <sup>b</sup>	4/4	406	NO
Calcium	20,000 - 150,000	28,000 - 41,000	116,000 <sup>b</sup>	4/4	70,250	NO
Iron	470 - 4,900	<100	1,000 <sup>c</sup>	4/4	2,405	YES

Not available.

EPA AWQC. Value presented is the Lowest Observed Effect Level (LOEL).

Based on the Lowest Chronic Value for All Organisms presented in Suter and Mabrey (1994).

EPA Ambient Water Quality Criteria, Fresh water chronic criteria.

**TABLE 3-1. SUMMARY OF CHEMICALS OF CONCERN: SURFACE WATER (CONTINUED)**

CHEMICALS OF CONCERN: CAPE LISBURNE INSTALLATION SURFACE WATER						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (µg/L)	BACKGROUND (µg/L)	ACTION LEVEL (µg/L)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (µg/L)	SELECTED AS COC
Magnesium	8,700 - 30,000	4,500 - 9,000	82,000 <sup>b</sup>	4/4	15,663	NO
Manganese	125 - 1,800	<50	1,100 <sup>b</sup>	3/4	670	YES
Sodium	18,500 - 38,000	4,000 - 5,600	680,000 <sup>b</sup>	4/4	26,125	NO

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS

CHEMICALS OF CONCERN: CAPE LISBURNE INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
<b>ORGANICS</b>						
DRPH	50 - 63,000	<60 - <150	500 <sup>a</sup>	17/50	4,464	YES
GRPH	1.03 - 150	<2 - <6	100 <sup>a</sup>	7/24	23.1	YES
RRPH	230 - 43,100	<120 - <300	2,000 <sup>a</sup>	13/50	2,133	YES
Benzene	0.05 - 0.26	<0.025 - <0.160	0.052 <sup>b</sup>	3/14	0.08	YES
Toluene	0.03 - 2.52	<0.025 - <0.160	0.786 <sup>c</sup>	9/14	0.43	YES
Ethylbenzene	0.07 - 0.69	<0.025 - <0.160	4.36 <sup>b</sup>	6/14	0.15	NO
Xylenes (total)	0.04 - 4.59	<0.05 - <0.32	1.21 <sup>b</sup>	9/14	0.82	YES
sec-Butylbenzene	0.19	<0.025 - <0.160	0.786 <sup>c</sup>	1/14	0.07	NO
1,4-Dichlorobenzene	0.1	<0.025 - <0.160	--	1/14	0.06	YES
Isopropylbenzene	0.15	<0.025 - <0.160	0.786 <sup>c</sup>	1/14	0.07	NO
p-Isopropyltoluene	0.16 - 0.6	<0.025 - <0.160	0.786 <sup>c</sup>	2/14	0.12	NO
Methylene chloride	0.07	<0.025 - <0.160	0.427 <sup>b</sup>	1/14	0.06	NO
Naphthalene	0.04 - 0.38	<0.025 - <0.160	0.407 <sup>b</sup>	2/14	0.08	NO

Not available.

ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991.

EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach summarized in Hull and Suter 1994).

EPA Sediment Quality Criteria for Toluene (see text).

EPA Sediment Quality Criteria for Ethylbenzene (see text).

EPA Sediment Quality Criteria for Aroclor 1254 (estimated in Hull and Suter 1994).

NOAA 1991, sediment ER-L (effects range-low).

Background for sodium represents background levels detected at eight Air Force arctic radar installations (Cape Lisburne background range of 58-120 mg/kg mistakenly categorizes sodium as a COC, and the sodium detected is probably due to ocean spray).

**TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS (CONTINUED)**

CHEMICALS OF CONCERN: CAPE LISBURNE INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
n-Propylbenzene	0.05 - 0.32	<0.025 - <0.160	0.786 <sup>c</sup>	3/14	0.09	NO
Trichloroethene	0.06 - 0.43	<0.025 - <0.160	1.07 <sup>b</sup>	2/14	0.09	NO
1,2,4-Trimethylbenzene	0.03 - 2.39	<0.025 - <0.160	4.36 <sup>d</sup>	9/14	0.54	NO
1,3,5-Trimethylbenzene	0.14 - 1.52	<0.025 - <0.160	4.36 <sup>d</sup>	7/14	0.26	NO
2-Methylnaphthalene	2.29 - 5.12	<0.250 - <4.23	--	2/5	3.41	NO
4-Methylphenol	7.32	<0.250 - <4.23	--	1/6	3.84	YES
Aroclor 1254	0.24 - 1,365	<0.2 - <0.3	0.17 <sup>b</sup>	3/50	27.96	YES
Aroclor 1260	0.02 - 6,290	<0.1 - 20	0.17 <sup>e</sup>	32/50	273.77	YES
<b>INORGANICS</b>						
Aluminum	3,850 - 7,400	4,700 - 17,000	--	4/4	5,613	NO
Barium	230 - 585	590 - 2,000	--	4/4	424	NO
Calcium	5,900 - 41,000	2,700 - 240,000	--	4/4	20,275	NO
Chromium	7.2 - 11.5	9.3 - 33	81 <sup>f</sup>	4/4	9	NO
Cobalt	4.7	<6.3 - 17	--	1/4	4	NO

Not available.

ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991.

EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach summarized in Hull and Suter 1994).

EPA Sediment Quality Criteria for Toluene (see text).

EPA Sediment Quality Criteria for Ethylbenzene (see text).

EPA Sediment Quality Criteria for Aroclor 1254 (estimated in Hull and Suter 1994).

NOAA 1991, sediment ER-L (effects range-low).

Background for sodium represents background levels detected at eight Air Force arctic radar installations (Cape Lisburne background range of 58-120 mg/kg mistakenly categorizes sodium as a COC, and the sodium detected is probably due to ocean spray).

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS (CONTINUED)

CHEMICALS OF CONCERN: CAPE LISBURNE INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
Copper	7.4 - 23	12 - 17	34 <sup>f</sup>	4/4	13	NO
Iron	8,100 - 16,000	5,400 - 39,000	--	4/4	12,063	NO
Magnesium	1,500 - 11,000	1,000 - 34,000	--	4/4	4,838	NO
Manganese	94 - 505	15 - 1,000	--	4/4	228	NO
Nickel	12 - 17	13 - 80	21 <sup>f</sup>	4/4	14	NO
Potassium	363 - 1,370	<540 - 2,600	--	3/4	689	NO
Sodium	104 - 290	<160 - 680 <sup>g</sup>	--	4/4	171	NO
Vanadium	16 - 27	19 - 58	--	4/4	19	NO
Zinc	43 - 73	40 - 250	150 <sup>f</sup>	4/4	58	NO

Not available.

ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991.

EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach summarized in Hull and Suter 1994).

EPA Sediment Quality Criteria for Toluene (see text).

EPA Sediment Quality Criteria for Ethylbenzene (see text).

EPA Sediment Quality Criteria for Aroclor 1254 (estimated in Hull and Suter 1994).

NOAA 1991, sediment ER-L (effects range-low).

Background for sodium represents background levels detected at eight Air Force arctic radar installations (Cape Lisburne background range of 58-120 mg/kg mistakenly categorizes sodium as a COC, and the sodium detected is probably due to ocean spray).

STEP TWO: Is the chemical detected above the action level or toxicity value?

**No:** Not considered a COC.  
**Yes:** Continue to step three.

STEP THREE: Is the chemical detected at a frequency greater than 5 percent?

**No:** Not considered a COC.  
**Yes:** Continue to step four.

STEP FOUR: Is the average concentration of the chemical greater than the maximum background concentration and is the maximum detected concentration greater than two times the maximum background concentration?

**No:** Not considered a COC.  
**Yes:** Chemical is classified as a COC.

All data for COCs were averaged (arithmetic mean) according to media. In the case of non-detects, averages were calculated using one-half of the quantitation limits. Replicate samples were averaged and treated as one sample. Total metal concentrations were used in determining COCs in surface water. This is a conservative approach because dissolved metal concentrations (the more bioavailable fraction) can be significantly lower than total metal concentrations. Section 3.1.1 describes surface water COCs, and Section 3.1.2 describes soil and sediment COCs. Any exceptions to the selection methodology are discussed in these sections.

### **3.1.1 Surface Water**

Analytical results from two sites, Spill/Leak #3 (ST07) and the Landfill and Waste Accumulation Area (LF01), were compiled and evaluated to identify the organic COCs in surface water. Analysis for inorganics (i.e., metals) was performed on the surface water samples collected from the Landfill and Waste Accumulation Area. Surface water samples were collected and analyzed for contaminants likely to be present at the specific sites. Not all samples were analyzed for a "full suite" of parameters, but instead were analyzed for some combination of the following: DRPH, GRPH, RRPH, BTEX, halogenated volatile organic compounds (HVOCs), VOCs, semivolatile organic compounds (SVOCs), PCBs, pesticides, and metals. Complete analytical results for all sampling conducted at the installation are presented in Appendix G. The following sections present the evaluation of the surface water data. Table 3-1 summarizes the evaluation and selection of COCs in surface water.

**3.1.1.1 Organic Compounds.** Eleven organic compounds were detected in surface water samples collected from the Cape Lisburne installation: benzene, toluene, ethylbenzene, xylenes (total), carbon tetrachloride, chloroform, cis-1,2-dichloroethene, naphthalene, tetrachloroethene, trichloroethene, and 1,2,4-trimethylbenzene. This section presents the evaluation of these compounds as COCs in surface water for the ERA.

**Benzene** was detected in two of six surface water samples at a concentration of 5 µg/L in both detections. Background sampling resulted in non-detects at a detection limit of 1 µg/L. Although the concentrations exceeded background levels, the detected concentration was well below the action level of 5,300 µg/L. As a result, benzene was not considered a COC.

**Toluene** was detected in one of six surface water samples at a concentration of 9.6 µg/L. Background level concentrations of toluene were not detected at a detection limit of 1 µg/L. Although the concentrations exceeded background levels, the detected concentration was well below the action level of 17,500 µg/L. As a result, toluene was not considered a COC.

**Ethylbenzene** was detected in two of six surface water samples at a concentration of 2 µg/L in both samples. Ethylbenzene was not detected in background samples at a detection limit of 1 µg/L. The action level for ethylbenzene is 32,000 µg/L, and because the sample concentration did not exceed the action level, ethylbenzene was not selected as a COC.

**Xylenes** were detected in three of six surface water samples at concentrations ranging from 4 to 5 µg/L. This compares to a background non-detect level of 2 µg/L and an action level of 62,308 µg/L. Because the sampled concentration was below the action level, xylene was not selected as a COC.

**Carbon tetrachloride** was detected in two of six surface water samples at concentrations of 24.7 and 89 µg/L. This compares to a background non-detect level of 1 µg/L and an action level of 35,200 µg/L. Because the sample concentrations were below the action level, carbon tetrachloride was not selected as a COC.

**Chloroform** was detected in two of six surface water samples at concentrations of 3.6 and 16 µg/L. This compares to a background non-detect level of 1 µg/L and an action level of 1,240 µg/L. Because the sample concentrations were below the action level, chloroform was not selected as a COC.

**cis-1,2-Dichloroethene** was detected in one of six surface water samples at a concentration of 1.35 µg/L. Background levels of cis-1,2-dichloroethene were not detected at a detection limit of 1 µg/L. Although the concentrations exceeded background levels, the detected concentration was well below the action level of 9,538 µg/L. As a result, cis-1,2-dichloroethene was not considered a COC.

**Naphthalene** was detected in one of six surface water samples at a concentration of 1.60 µg/L. Background level concentrations of naphthalene were not detected at detection limits of 1 µg/L. Although the concentrations exceeded background levels, the detected concentration was well below the action level of 620 µg/L. As a result, naphthalene was not considered a COC.

**Tetrachloroethene** was detected in one of six surface water samples at a concentration of 4 µg/L. Background levels of tetrachloroethene were not detected at detection limits of 1 µg/L. Although the concentrations exceeded background levels, the detected concentration was well below the action level of 750 µg/L. As a result, tetrachloroethene was not considered a COC.

**Trichloroethene** was detected in two of six surface water samples at concentrations of 5.5 and 62 µg/L. Background levels of trichloroethene were not detected at detection limits of 1 µg/L. Although the concentrations exceeded background levels, the detected concentration was well below the action level of 21,900 µg/L. As a result, trichloroethene was not considered a COC.

**1,2,4-Trimethylbenzene** was detected in one of six surface water samples at a concentration of 1.15 µg/L. Background levels of 1,2,4-trimethylbenzene were not detected at detection limits of 1 µg/L. There is no action level currently established for 1,2,4-trimethylbenzene. As a result, 1,2,4-trimethylbenzene was selected as a COC, and the average concentration of 0.61 µg/L was used in the risk assessment.

**3.1.1.2 Metals.** Four surface water samples collected from the Landfill and Accumulation Area (LF01) were analyzed for metals. Seven inorganic analytes were detected: aluminum, barium, calcium, iron, magnesium, manganese, and sodium. This section presents the evaluation of these metals as COCs for the ERA. Analytes not detected in surface water samples were: antimony, arsenic, beryllium, cadmium, chromium, cobalt, copper, lead, molybdenum, nickel, potassium, selenium, silver, thallium, vanadium, and zinc. It is important to note that, in some cases, sample quantitation limits for certain metals were somewhat higher than ecologically relevant action levels. For example, in the case of copper, the sample quantitation limit was 50 µg/L. However, the current AWQC (based on a hardness value of 100 mg/L CaCO<sub>3</sub>) for copper is 12 µg/L. As a result, an ecological risk may exist for aquatic organisms from exposure to certain metals at their sample quantitation limits. These metals include: cadmium, chromium, copper, lead, and selenium. These issues will be addressed further in Section 3.5, Uncertainty Analysis. All concentrations of metals discussed below are results from total metal analyses.

**Aluminum** was detected in the two of four surface water samples at concentrations of 140 and 160 µg/L. Background concentrations were not detected at a detection level of 100 µg/L. The action level for aluminum is 460 µg/L, based on the "Lowest Chronic Value for All Organisms" (Suter and Mabrey 1994). Aluminum was not present in surface water at concentrations in excess of the action level, so it was not selected as a COC.

**Barium** was detected in all four surface water samples at concentrations ranging from 77 to 860 µg/L. Background concentrations of barium ranged from 79 - 92 µg/L. The action level for barium is 5,800 µg/L, based on data presented in Suter and Mabrey (1994). The value selected as the action level is the "Lowest Chronic Values for All Organisms". Barium was not selected as a COC because it did not exceed the action level.

**Calcium** was detected in all four surface water samples at concentrations ranging from 20,000 to 150,000 µg/L. Background concentrations ranged from 28,000 - 41,000 µg/L. The action level is 116,000 µg/L based on the "Lowest Chronic Value for All Organisms" (Suter and Mabrey 1994). Although one sample exceeded the action level, calcium was not selected as a COC because the sample location with the 150,000 µg/L detection was directly downgradient of a gravel pad composed of calcium-bearing rock. In addition, calcium is an essential nutrient, the concentrations of which are regulated by normal metabolic activity.



**Iron** was detected in all four surface water samples at concentrations ranging from 470 to 4,900 µg/L. Background concentrations were not detected at a detection limit of 100 µg/L. Iron concentrations exceed the background concentration in surface water and the action level of 1,000 µg/L (AWQC), so iron was selected as a COC. The average concentration used for evaluation in this ERA was 2,405 µg/L.

**Magnesium** was detected in all four surface water samples at concentrations ranging from 8,700 to 30,000 µg/L. Background concentrations ranged from 4,500 - 9,000 µg/L. There are no AWQC for magnesium. However, 82,000 µg/L may be used as a toxicological screening benchmark based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Magnesium was not selected as a COC in the ERA because it was below the action level.

**Manganese** was detected in three of four surface water samples at concentrations ranging from 730 to 1,800 µg/L. Background concentrations were not detected at a detection limit of 50 µg/L. The action level for manganese is 80.3 µg/L based on the Secondary Chronic Value presented in Suter and Mabrey (1994). Because manganese was detected at concentrations exceeding background and action levels, it was selected as a COC. The average concentration used for evaluation in this ERA was 670 µg/L.

**Sodium** was detected in all four surface water samples at concentrations ranging from 18,500 to 38,000 µg/L, above the background concentrations of 4,000 to 5,600 µg/L. The action level for sodium of 680,000 µg/L was based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Because onsite sodium concentrations at the Cape Lisburne installation did not exceed the action level, sodium was not selected as a COC.

### 3.1.2 Soils and Sediments

Soil/sediment sample analytical results from the five sites were compiled and evaluated to determine the COCs. Because most ecological receptors are principally exposed to surficial soils, only samples collected from 0 to 1.5 feet were considered in this ERA. Fifty soil/sediment samples were evaluated to determine the COCs. These samples were analyzed for contaminants likely to be present at the specific sites. Not all samples were analyzed for a "full suite" of parameters, but instead were analyzed for some combination of the following: DRPH, GRPH, RRPB, BTEX, HVOCS, VOCs, SVOCs, PCBs, pesticides, and metals. A summary of analytical results for all sampling conducted at the installation is presented in Appendix G. The following sections present the evaluation of the soil/sediment data for the five sites. Only compounds that were detected on the sites are discussed. Table 3-2 summarizes the evaluation and selection of COCs in soil and sediment.

**3.1.2.1 Petroleum Hydrocarbons.** Fifty soil/sediment samples were collected from the sites and selectively analyzed for DRPH and RRPB. GRPH was analyzed in 24 soil/sediment samples. A discussion of these petroleum hydrocarbon mixtures and their toxicity is presented in Section 3.3.1.

**DRPH** were detected in 17 of 50 soil/sediment samples at concentrations ranging from 50 to 63,000 mg/kg. DRPH were not detected in background samples at detection limits of 60 to 150

mg/kg. The action level for DRPH in soils/sediments is 500 mg/kg. Because DRPH were detected at levels above the action level, they were selected as a COC. The exposure concentration used in the risk assessment was the average concentration of 4,464 mg/kg.

**GRPH** were detected in 7 of 24 soil/sediment samples at concentrations ranging from 1.03 to 150 mg/kg. GRPH was not detected in background samples. The action level for GRPH is 100 mg/kg. GRPH were detected at concentrations above the action level, so they were considered a COC. GRPH were evaluated in the risk assessment as components of DRPH. Refer to Section 3.3.1 for a discussion of the toxicity of petroleum hydrocarbon mixtures.

**RRPH** were detected in 13 of 50 soil/sediment samples at concentrations ranging from 230 to 43,100 mg/kg. RRPH was not detected in background samples. The action level for RRPH is 2,000 mg/kg. Because RRPH were detected at concentrations above the action level, they were considered a COC. RRPH were evaluated in the risk assessment as components of DRPH. Refer to Section 3.3.1 for a discussion of the toxicity of petroleum hydrocarbon mixtures.

**3.1.2.2 Benzene, Toluene, Ethylbenzene, and Xylenes.** BTEX were analyzed using the 8020/8020 modified method and the VOC (8260) method. The data presented here and in Table 3-2 are the result of the VOC analyses (8260 method) because of a higher confidence level in the data (more consistent and refined detection limits). This approach is expected to be protective of ecological receptors. The following paragraphs summarize the analytical results.

**Benzene** was detected in 3 of 14 soil/sediment samples at concentrations ranging from 0.05 to 0.26 mg/kg. Benzene was not detected in background samples ( $<0.025$  -  $<0.160$  mg/kg). The estimated action level for benzene is 0.052 mg/kg, based on the equilibrium partitioning approach presented in Hull and Suter (1994). Because concentrations of benzene exceeded the action level, benzene was considered a COC at the Cape Lisburne installation. The average concentration at the Cape Lisburne sites was 0.08 mg/kg.

**Toluene** was detected in 9 out of 14 soil/sediment samples at concentrations ranging from 0.03 to 2.52 mg/kg. Toluene was not detected in background samples at quantitation limits of 0.025 to 0.160 mg/kg. The action level for this compound is 0.786 mg/kg. Because concentrations at the site exceeded the action level, toluene was selected as a COC. The average concentration at the Cape Lisburne sites was 0.43 mg/kg.

**Ethylbenzene** was detected in 6 of 14 soil/sediment samples at concentrations ranging from 0.07 to 0.69 mg/kg. Ethylbenzene was not detected in background samples at quantitation limits of 0.025 to 0.160 mg/kg; the action level is 4.36 mg/kg, based on the equilibrium partitioning approach presented in Hull and Suter (1994). Because onsite concentrations did not exceed action levels, ethylbenzene was not considered a COC.

**Xylenes (total)** were detected in 9 of 14 soil/sediment samples. Xylene concentrations ranged from 0.04 to 4.59 mg/kg. Xylenes were not detected in background samples at quantitation limits of 0.05 to 0.32 mg/kg. The action level is 1.21 mg/kg. Xylenes were considered a COC because onsite concentrations were above action levels. The exposure concentration used in this ERA was the average concentration of 0.82 mg/kg.

**3.1.2.3 Volatile Organic Compounds.** In addition to the BTEX, ten VOCs were detected in soil/sediment samples collected and analyzed using the 8260 method. The compounds detected were: sec-butylbenzene, 1,4-dichlorobenzene, isopropylbenzene, p-isopropyltoluene, methylene chloride, naphthalene, n-propylbenzene, trichloroethene, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene. None of these chemicals was detected in background samples (quantitation limits of 0.025 to 0.160 mg/kg). This section presents an evaluation of these compounds as COCs for the ERA.

Toluene, ethylbenzene, sec-butylbenzene, isopropylbenzene, p-isopropyltoluene, n-propylbenzene, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene are alkylbenzenes, a class of chemicals typically found in fuel oils (i.e., DRPH/GRPH) (ATSDR 1993). In order to evaluate the toxicity and potential for selection as COCs of those chemicals that do not have action levels (all the listed alkylbenzenes with the exception of toluene and ethylbenzene), comparisons to the toxicities of toluene and ethylbenzene are made. The rationale and methodology for these comparisons follow.

Generally, as functional groups (e.g., methyl-, ethyl-, butyl-) are added to a benzene ring, the octanol water partition coefficient ( $K_{ow}$ ), which controls the bioavailability of nonpolar organic compounds (Baudo et al. 1990), increases. In addition, as the number of functional groups increase, the toxicity of the chemical tends to increase. However, compounds with a large  $K_{ow}$  are more tightly sorbed to sediments and thus less bioavailable. This decrease in bioavailability offsets the increase in toxicity. Thus, the sediment screening values also increase as functional groups are added and as  $K_{ow}$  increases. This trend may be observed in the sediment screening values: benzene, 0.052 mg/kg; toluene, 0.786 mg/kg; and ethylbenzene, 4.36 mg/kg (Hull and Suter 1994). The log  $K_{ow}$ s of benzene (2.13), toluene (2.69), and ethylbenzene (3.15) illustrate the direct relationship between the  $K_{ow}$ , toxicity, and the sediment screening values. Examining the  $K_{ow}$  of alkylbenzenes illustrates an important point: as  $K_{ow}$  increases, the expected toxicity in sediments declines. Table 3-3 presents the log  $K_{ow}$ s and estimated sediment quality criteria for benzene, toluene, and ethylbenzene.

**sec-Butylbenzene** was detected in 1 of 14 soil/sediment samples at a concentration of 0.19 mg/kg. There are no action levels available for this compound. However, sec-butylbenzene is an alkylbenzene and may be compared with the toxicity of toluene. The log  $K_{ow}$  for sec-butylbenzene is 4.24. Based on the information presented above, it appears that sec-butylbenzene is less toxic than toluene. The action level for toluene is 0.786 mg/kg, and its average concentration is 0.43 mg/kg. The average concentration of sec-butylbenzene is 0.07 mg/kg, below that of toluene, so the evaluation of toluene will serve to represent the risk estimate for sec-butylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection), it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes (constituents of DRPH).

**TABLE 3-3. CHEMICAL/PHYSICAL PROPERTIES**

Chemical	Log K <sub>ow</sub> <sup>a</sup>	Sediment Quality Criteria (mg/kg) <sup>b</sup>
Benzene	2.13	0.052
Toluene	2.69	0.786
Ethylbenzene	3.15	4.36

<sup>a</sup> EPA 1992c.

<sup>b</sup> Hull and Suter 1994.

**1,4-Dichlorobenzene** was detected in 1 of 14 soil/sediment samples at a concentration of 0.1 mg/kg. There are no action levels available for this compound, so it was selected as a COC.

**Isopropylbenzene** was detected in 1 of 14 soil/sediment samples from the Cape Lisburne sites at a concentration of 0.15 mg/kg. There are no action levels available for this chemical. However, isopropylbenzene is an alkylbenzene and may be compared with the toxicity of toluene. The log K<sub>ow</sub> of isopropylbenzene is 3.66. Based upon the discussion above, the toxicity of isopropylbenzene is likely to be less than that of toluene. The action level for toluene is 0.786 mg/kg, and its average concentration is 0.43 mg/kg. The average concentration of isopropylbenzene is 0.07 mg/kg, below that of toluene, so the evaluation of toluene will serve to represent the risk estimate for isopropylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes (constituents of DRPH).

**p-Isopropyltoluene** (also an alkylbenzene) was detected in 2 of 14 soil/sediment samples at concentrations of 0.16 and 0.6 mg/kg. There are no action levels for this compound. The log K<sub>ow</sub> of p-isopropyltoluene is 4.10. Based on the discussion above, it is expected that p-isopropyltoluene is less toxic than toluene because the log K<sub>ow</sub> of p-isopropyltoluene is greater than that of toluene. The action level for toluene is 0.786 mg/kg, and its average concentration is 0.43 mg/kg. The average concentration of p-isopropyltoluene was 0.12 mg/kg, below that of toluene, so the evaluation of toluene will serve to represent the risk estimate for p-isopropyltoluene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes (constituents of DRPH).

**Naphthalene** was detected in 2 of 14 soil/sediment samples at concentrations of 0.04 and 0.38 mg/kg. The action level for this compound is 0.407 mg/kg. Because it was not detected above the action level, naphthalene was not selected as a COC.

**n-Propylbenzene** was detected in 3 of 14 soil/sediment samples at concentrations ranging from 0.05 to 0.32 mg/kg. There are no action levels for this compound, which is an alkylbenzene. The  $\log K_{ow}$  for n-propylbenzene is 3.68. Based on the discussion above, n-propylbenzene is likely to be less toxic than toluene because the  $\log K_{ow}$  of n-propylbenzene is greater than that of toluene. The action level for toluene is 0.786 mg/kg, and its average concentration is 0.43 mg/kg. The average concentration of n-propylbenzene was 0.09 mg/kg, below that of toluene, so the evaluation of toluene will serve to represent the risk estimate for n-propylbenzene. In addition, because DRPH are a COC (detected at higher concentrations and higher frequencies of detection) it is assumed that the evaluation of the toxicity of DRPH will conservatively account for the incremental risk associated with low levels of alkylbenzenes (constituents of DRPH).

**Trichloroethene** was detected in 2 of 4 soil/sediment samples at concentrations of 0.06 and 0.43 mg/kg. The action level for trichloroethene is 1.07 mg/kg (Hull and Suter 1994). Trichloroethene was not considered a COC because it did not exceed the action level.

**1,2,4-Trimethylbenzene** was detected in 9 of 14 soil/sediment samples at concentrations ranging from 0.03 to 2.39 mg/kg. There is no action level for this compound. 1,2,4-Trimethylbenzene is classified as an alkylbenzene. The  $\log K_{ow}$  for 1,2,4-trimethylbenzene is 3.63. Based on the discussion presented above, 1,2,4-trimethylbenzene is likely to be less toxic than both toluene and ethylbenzene because the  $\log K_{ow}$  of 1,2,4-trimethylbenzene is greater than that of toluene and ethylbenzene. The maximum concentration of 1,2,4-trimethylbenzene was 2.39 mg/kg, which is less than the action level for ethylbenzene of 4.36 mg/kg. As a result, 1,2,4-trimethylbenzene was not selected as a COC.

**1,3,5-Trimethylbenzene** was detected in 7 of 14 soil/sediment samples at concentrations ranging from 0.14 to 1.52 mg/kg. There is no action level for this compound. 1,3,5-Trimethylbenzene is also classified as an alkylbenzene. The  $\log K_{ow}$  for 1,3,5-trimethylbenzene is 3.42. Based on the discussion presented above, 1,3,5-trimethylbenzene is likely to be less toxic than both toluene and ethylbenzene because the  $\log K_{ow}$  of 1,3,5-trimethylbenzene is greater than that of toluene and ethylbenzene. The maximum concentration of 1,3,5-trimethylbenzene was 1.52 mg/kg, which is less than the action level for ethylbenzene of 4.36 mg/kg. As a result, 1,3,5-trimethylbenzene was not selected as a COC.

**3.1.2.4 Semivolatile Organic Compounds.** Two semivolatile organic compounds were detected at the Cape Lisburne sites: 2-methylnaphthalene and 4-methylphenol. These chemicals were not detected in background samples at quantitation limits ranging from 0.311 to 8.2 mg/kg. The quantitation limits for SVOCs ranged as high as 29 mg/kg. Because incorporating non-detects (using one-half the quantitation limit) with unusually high quantitation limits skews the average (artificially inflating the average above the maximum detected concentration), samples with quantitation limits that were more than twice the maximum detection were not considered in the COC analysis. Thus, four 2-methylnaphthalene non-detect samples, with quantitation limits of 11.8, 18.5, 20.9, and 29 mg/kg, and three 4-methylphenol non-detect samples, with quantitation limits of 18.5, 20.9, and 29 mg/kg, were eliminated from the COC analysis.

**2-Methylnaphthalene** was detected in two of five soil/sediment samples at concentrations of 2.29 and 5.12 mg/kg. There are no action levels available for this compound. Naphthalenes are

constituents of fuel oils (ATSDR 1993). As a result, it is assumed that the evaluation of the toxicity of DRPH (detected both at higher concentrations and at a higher frequency of detection) will conservatively account for the incremental risk associated with 2-methylnaphthalene.

**4-Methylphenol** was detected in one of six soil/sediment samples at a concentration of 7.32 mg/kg. There are no action levels available for this compound, and it is not a common constituent of fuel oils. Therefore, it was selected as a COC. The average concentration used in the risk assessment is 3.84 mg/kg.

**3.1.2.5 Polychlorinated Biphenyls.** Fifty soil/sediment samples from the Cape Lisburne sites were analyzed for PCBs. Only two of the eight Aroclor groups of PCBs analyzed were detected.

**Aroclor 1254** was detected in 3 of 50 soil/sediment samples at concentrations ranging from 0.24 to 1,365 mg/kg. The action level for Aroclor 1254 is 0.17 mg/kg (Hull and Suter 1994). Aroclor 1254 was selected as a COC because its concentrations exceeded the action level.

**Aroclor 1260** was detected in 32 of 50 soil/sediment samples at concentrations ranging from 0.02 to 6,290 mg/kg. The action level for Aroclor 1254 (0.17 mg/kg) was used to screen for Aroclor 1260. As a result, Aroclor 1260 was selected as a COC because it exceeded the action level.

**3.1.2.6 Metals.** Fourteen inorganic analytes were detected in four soil/sediment samples collected. The metals detected were: aluminum, barium, calcium, chromium, cobalt, copper, iron, magnesium, manganese, nickel, potassium, sodium, vanadium, and zinc. This section presents the evaluation of these metals as COCs for the ERA.

**Aluminum** was detected in all four soil/sediment samples. Concentrations ranged from 3,850 to 7,400 mg/kg. Background concentrations ranged from 4,700 to 17,000 mg/kg. There is no action level for aluminum. Because onsite concentrations did not exceed background concentrations, aluminum was not selected as a COC.

**Barium** was detected in all four soil/sediment samples analyzed for metals at concentrations between 230 and 585 mg/kg. The background concentrations of barium ranged from 590 to 2,000 mg/kg. There is no action level for barium. Because onsite concentrations did not exceed background concentrations, barium was not selected as a COC.

**Calcium** was detected in all four soil/sediment samples. Concentrations ranged from 5,900 to 25,000 mg/kg. Background concentrations ranged from 2,700 to 240,000 mg/kg. There is no action level for calcium. Because onsite concentrations did not exceed background concentrations, calcium was not selected as a COC.

**Chromium** was detected in all four soil/sediment samples. Concentrations ranged from 7.2 to 11.5 mg/kg. The maximum background concentration was 33 mg/kg. The action level for chromium is 81 mg/kg (NOAA 1991). Because the detected concentrations did not exceed the background levels, chromium was not selected as a COC.

**Cobalt** was detected in one of four soil/sediment samples at a concentration of 4.7 mg/kg. The maximum background concentration was 17 mg/kg. No action level is available for cobalt, but because the detected concentrations did not exceed the background level, cobalt was not selected as a COC.

**Copper** was detected in all four soil/sediment samples collected from the Cape Lisburne installation. Detected concentrations ranged from 7.4 to 23 mg/kg. Background concentrations ranged from 12 to 17 mg/kg. The action level for copper is 34 mg/kg (NOAA 1991). Because the detected concentrations did not exceed the background levels, copper was not selected as a COC.

**Iron** was detected in all four soil/sediment samples. Concentrations ranged from 8,100 to 16,000 mg/kg. The background concentrations ranged from 5,400 to 39,000 mg/kg. There is no action level for iron. Because the detected concentrations did not exceed background concentrations, iron was not selected as a COC.

**Magnesium** was detected in all four soil/sediment samples. Concentrations ranged from 1,500 to 11,000 mg/kg. The background concentrations for magnesium ranged from 1,000 to 34,000 mg/kg. There is no action level for magnesium. Because magnesium was not detected above background concentrations, it was not selected as a COC.

**Manganese** was detected in all four soil/sediment samples. Concentrations ranged from 94 to 505 mg/kg. The background concentrations for manganese ranged from 15 to 1,000 mg/kg. There are no action levels for manganese. Because onsite concentrations did not exceed background concentrations, manganese was not selected as a COC.

**Nickel** was detected in all four soil/sediment samples ranging in concentration from 12 to 17 mg/kg. The background concentrations ranged from 13 to 80 mg/kg. The action level for nickel is 21 mg/kg (NOAA 1991). Nickel was not selected as a COC because onsite concentrations did not exceed the background levels.

**Potassium** was detected in three of four soil/sediment samples at concentrations ranging from 363 to 1,370 mg/kg. The background concentrations ranged from <540 to 2,600 mg/kg. There is no action level for potassium. This metal was not selected as a COC because onsite concentrations were below background concentrations.

**Sodium** was detected in all four soil/sediment samples in soil/sediment. Concentrations detected ranged from 104 to 290 mg/kg. There is no action level for sodium in soil/sediment. The background concentrations at the Cape Lisburne installation ranged from 58 to 120 mg/kg, and the background concentrations from eight Air Force arctic radar installations were between <160 and 680 mg/kg. Sodium is not typically considered to be a toxic element, thus the comparison to the Cape Lisburne background data, which results in classifying sodium as a COC, was not used. Rather, the comparison was made to the 680 mg/kg maximum background concentration. Because the detected concentrations did not exceed 680 mg/kg, sodium was not selected as a COC.

**Vanadium** was detected in all four soil/sediment samples, ranging in concentration from 16 to 27 mg/kg. The background concentrations ranged from 19 to 58 mg/kg. There is no action level for vanadium. This metal was not selected as a COC because onsite concentrations were below background concentrations.

**Zinc** was detected in all four soil/sediment samples at concentrations ranging from 43 to 73 mg/kg. The background concentrations for zinc ranged from 40 to 250 mg/kg. The action level for zinc is 150 mg/kg (NOAA 1991). Because zinc was not detected above the action or background levels, it was not selected as a COC.

### **3.2 ECOLOGICAL EXPOSURE ASSESSMENT**

The vegetation of the Arctic Coastal Plain and the ecosystems it characterizes have developed primarily as a result of the low relief and harsh environment. The growing season is short, typically extending from June through mid-September. Winters are long, cold, dry, and dark. Air temperatures that average below freezing for most of the year result in a permafrost layer that begins near the surface and reaches to depths as great as 610 meters. Seasonal thawing results in an active layer between ground surface and 3.7 meters below the surface (Hart Crowser 1987).

The impervious permafrost layer prevents percolation and infiltration of water below the active layer, and the generally flat terrain provides poor drainage. As a result, the ecosystems of the Arctic Coastal Plain are often defined not only by their plant associations but also by the degree of water found in and on them. Hart Crowser (1987) describes five major ecosystems for the classification of tundra and Arctic Coastal Plain communities:

- **Marine zones:** these include lagoons, estuaries, barrier islands, strands, and beaches. The abundance of vegetation along the marine coastal zone is inversely related to the amount of beach scouring by waves and ice. Mainland beaches support a variety of vegetation, including sedges, grasses, and forbs.
- **Wet sedge meadows:** an association of meadows, ponds, and lakes also known as "wet tundra". This system, with its associated wetlands, is dominant in the area extending west from the Colville River to the Chukchi Sea (including the Point Lonely, Point Barrow, Wainwright, Point Lay, and Cape Lisburne installations). Differences in vegetation within this ecosystem are related to moisture and microrelief.
- **Tussock tundra:** or "moist tundra" consisting primarily of areas dominated by tussock-forming cottongrass. This system covers significant portions of the Arctic Coastal Plain.
- **Riverine systems and floodplains:** including riparian shrubland on recent and old alluvium. Being better drained than surrounding lands, the riparian environment supports a distinctive "shrub thicket" vegetation.



- Alpine tundra: including rocky upland areas of sparse mat-forming or fell-field vegetation.

The species associated with each ecosystem at the Cape Lisburne radar installation have the potential to be exposed to COCs if exposure pathways are complete. If pathways are complete, the representative species selected are considered potential receptors. Figure 3-1 presents a schematic model of the potential exposure pathways.

The Ecological Exposure Assessment segment of the risk assessment lists the most common species found at Air Force arctic radar installations in Section 3.2.1, species of the Arctic Coastal Plain; the representative species and the rationale used for their selection in Section 3.2.2; a discussion of the exposure pathways in Section 3.2.3; and a review of the habitat suitability for representative species in Section 3.2.4. Sections 3.2.5, 3.2.6, and 3.2.7 provide the methodology of the exposure assessment for representative plants, aquatic species, and birds and mammals, respectively. Life history tables, which provide species specific information for use in the exposure assessment, are included in Section 3.2.7.

### 3.2.1 Species of the Arctic Coastal Plain

The representative species used in the ERA for the Cape Lisburne installation were selected from species characteristic of all the Air Force radar installations along the Arctic Coastal Plain and are detailed in Sections 3.2.2.1 through 3.2.2.5.

The Cape Lisburne installation is located along the northwestern boundary of the Arctic Coastal Plain. Hart Crowser (1987) and Woodward-Clyde (1993) have listed the species likely to occur along the Coastal Plain based on site-specific studies and a review of the literature. The marine zone, wet sedge meadows, tussock tundra, and alpine tundra are the primary ecosystems found at the Cape Lisburne installation. Site-specific surveys of the ecosystems associated with the Air Force arctic radar installations have not been conducted for this risk assessment; however, a study investigating the abundance and distribution of Steller's and spectacled eiders was used (Alaska Biological Research 1994).

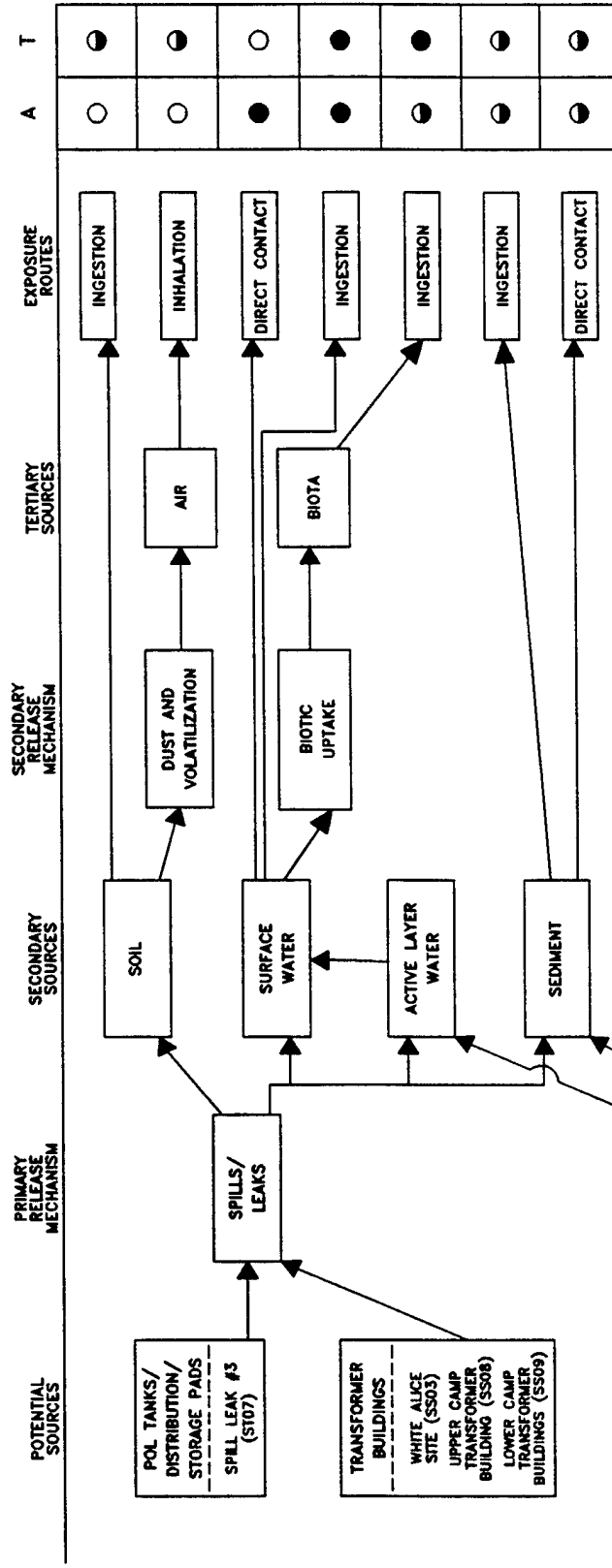
**3.2.1.1 Plants.** Plants commonly associated with the marine zone are sedges, grasses, and forbs. *Carex subspathacea* and *C. aquatilis* are dominant plants in the coastal wetlands.

The wet sedge meadow (also known as "wet tundra") is characterized by a variety of sedges and grasses. Typical species include: cottongrass, *Eriophorum* spp.; tundra grass, *Dupontia fischeri*; and mosses, *Sphagnum* spp. Marsh marigold, *Caltha palustris*, and horsetail, *Equisetum* spp. may be found in wetter areas (Hart Crowser 1987).

The tussock tundra (or moist tundra) is drier than the wet sedge meadow/wet tundra association. Tussock-forming cottongrass is the dominant plant species. Grasses, sedges, dwarf shrubs, mosses, and lichens are scattered throughout the tussock complex. These species include: willows, *Salix* spp.; Labrador tea, *Ledum palustre*; blueberry and lingonberry, *Vaccinium* spp.; and lousewort, *Pedicularis* spp. (NPRA Task Force 1978; Bergman et al. 1977).

THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. LIS-FLO2



**CAPE LISBURNE  
RADAR INSTALLATION**

**USAF 611th CES**

FIGURE NO. 3-1

**ECOLOGICAL RISK  
ASSESSMENT  
POTENTIAL EXPOSURE  
PATHWAYS**

- A AQUATIC RECEPTORS
- T TERRESTRIAL RECEPTORS
- COMPLETE EXPOSURE PATHWAY
- POTENTIALLY COMPLETE PATHWAY
- INCOMPLETE OR INSIGNIFICANT EXPOSURE PATHWAY

THIS PAGE INTENTIONALLY LEFT BLANK

Riverine/riparian systems are composed of a diversity of habitat types and species. The dominant plants are shrubs with a scattered understory of grasses and herbs. Larkspur, *Delphinium brachycentrum*; cinquefoil, *Potentilla* spp.; bearberry, *Arctostaphylos* spp.; and wormwood, *Artemisia arctica* are common species (NPRA Task Force 1978; Bergman et al. 1977).

Alpine tundra is found at higher elevations that are better drained than the wet and moist tundras. This community is characterized by lichens, forbs, and woody shrubs. Bearberry, Labrador tea, dwarf birch, and *Salix* spp. are common species (Hart Crowser 1987).

**3.2.1.2 Aquatic Organisms.** Sixty-six species of fish inhabiting marine, estuarine, and freshwater systems have been identified in the arctic region (Hart Crowser 1987). Marine species inhabiting the nearshore and offshore waters include: boreal smelt, *Osmerus eperlanus*; Pacific herring, *Clupea harengus*; arctic cod, *Boreogadus saida*; and fourhorn sculpin, *Myoxocephalus quadricornis*. Anadromous species using arctic rivers for spawning include the arctic cisco, *Coregonus autumnalis*; arctic char, *Salvelinus alpinus*; and occasional pink and chum salmon, *Onchorhynchus* spp. Lack of overwintering habitat is a significant limiting condition for both anadromous and freshwater fish of the arctic region. The principal freshwater fish found in the region are grayling, *Thymallus arcticus*; lake trout, *Salvelinus namaycush*; burbot, *Lota lota*; and nine-spined stickleback, *Pungitius pungitius* (Hart Crowser 1987).

Invertebrates that may be present in the waters and wet habitats of the Arctic Coastal Plain are well represented by the crustaceans (i.e., copepods, isopods, amphipods, and decapods).

**3.2.1.3 Birds.** There are approximately 180 species of birds seasonally associated with the habitats of the Arctic Coastal Plain. Of these, many are shorebirds and waterfowl using migratory corridors that pass through the Cape Lisburne area (U.S. Air Force 1993). The rock cliffs approximately one mile west of the Cape Lisburne installation are important nesting areas for black-legged kittiwakes, *Rissa tridactyla*, and common and thick-billed murres *Uria* spp. Bird use of the coastal plain is highly seasonal and associated with typical avian breeding and migration cycles. Shoreline habitats are used significantly in association with molting, pre-migratory staging, and post breeding movement. These habitats are considered critical by the U.S. Fish and Wildlife Service [U.S. Fish and Wildlife Service (USFWS) 1982]. Principal species include: glaucous gull, *Larus hyperboreus*; red phalarope, *Phalaropus fulicaria*; dunlin, *Calidris alpina*; loons, *Gavia* spp.; sandpipers, *Calidris* spp.; eiders, *Somateria* spp.; and geese, *Branta* spp. and *Chen* spp. Among the migratory passerine species using the coastal habitats are the Savannah sparrow, *Passerculus sandwichensis*; common and hoary redpolls, *Carduelis* spp.; snow bunting, *Plectrophenax nivalis*; and Lapland longspur, *Calcarius lapponicus* (Woodward-Clyde 1993).

**3.2.1.4 Mammals.** The mammalian fauna of the Arctic Coastal Plain and adjacent waters is relatively simple compared to fauna at lower latitudes. A review of species lists indicates a total of 38 species that commonly occur in the arctic; 11 of these are marine mammals (Hart Crowser 1987). A sampling of the terrestrial mammals geographically associated with the Air Force arctic radar stations, including Cape Lisburne, consists of brown lemming, *Lemmus trimucronatus*; masked shrew, *Sorex cinereus*; arctic fox, *Alopex lagopus*; red fox, *Vulpes vulpes*;

weasels, *Mustela* spp.; tundra vole, *Microtus oeconomus*; caribou, *Rangifer tarandus*; and grizzly bear, *Ursus arctos* (Hart Crowser 1987; Woodward-Clyde 1993).

Marine mammals of the arctic coast include polar bear, *Ursus maritimus*; walrus, *Odobenus rosmarus*; six species of whales; and five species of seals. The most common of the whale and seal species are beluga whale, *Delphinapterus leucas*; bowhead whale, *Balaena mysticetus*; gray whale, *Eschrichtius robustus*; ringed seal, *Phoca hispida*; and bearded seal, *Erignathus barbatus* (Hensel et al. 1984).

**3.2.1.5 Threatened and Endangered Species.** Species of the Arctic Coastal Plain and nearby waters that are protected by federal and state designations include bowhead whale (endangered); fin whale, *Balaenoptera physalus* (endangered); sei whale, *Balaenoptera borealis* (endangered); and hump-backed whale, *Megaptera novaengliae* (endangered). The gray whale was delisted by the National Marine Fisheries Service 16 June 1994. Avian species include the spectacled eider, *Somateria fischeri* (threatened), and Steller's eider, *Polysticta stelleri* (candidate for threatened status). Based on the latest federal and state lists of threatened and endangered plant species (June 1995), no plant species at the Air Force arctic radar installations are currently listed.

### **3.2.2 Representative Species**

It is impractical to evaluate all of these potential receptors individually because of the great diversity of plants and animals at a given site. Thus, for ERAs, a set of "representative species" is selected for further evaluation. The representative species are selected primarily on the species' likelihood of exposure based on their preferred habitat, feeding habits, and distribution of contaminants. Potential exposure pathways are shown in Figure 3-1 and discussed in Section 3.2.3. The abundance of a species, relative to the areal extent of the sites, is also considered. The representative species encompass a range of ecological niches in order to achieve the best characterization of the ecosystems being examined. In addition, species are selected, in part, as a result of the availability of toxicity, exposure, and life history information. Species that may be sensitive to environmental impacts, such as endangered or threatened species, are also evaluated. Any endangered or threatened species discussed in the ERA are not considered representative of the Arctic Coastal Plain or the Air Force arctic radar installations. These species are evaluated to provide information about whether they face potential risks from exposure to the COCs being evaluated in the ERA.

For the Air Force arctic radar stations, groups of receptors are evaluated, including plants, aquatic invertebrates, fish, birds, and mammals. Potential risks to representative species are estimated by evaluating sampling data for the relevant exposure media (i.e., soil, sediment, and surface water). For plants, soil/sediment COC data are used to estimate potential uptake; for aquatic species, surface water COC concentrations are used to estimate exposure. For the birds and mammals selected, exposures are estimated by evaluating their potential dietary intakes of COCs. No site-specific studies were conducted to determine exposure or toxicity levels at the installation.

The similarity of ecosystems at each of the installations allows the use of the same set of representative species for all installations. It may be possible that a representative species inhabits the general area of an installation, but does not occur specifically on the installation property. When and if this situation occurs, it will be noted. The receptors that are selected specifically as representative species for the Cape Lisburne installation are listed in the paragraphs that discuss the representative groups (i.e., plants, aquatic organisms, birds, mammals, and threatened or endangered species). Table 3-4 presents the representative and sensitive species for Air Force arctic radar installations, including endangered and threatened species that may be potentially exposed. The USFWS was consulted about the occurrence and selection of threatened and endangered species.

**3.2.2.1 Representative Plants.** Plants selected as representative species include: sedges; willows; cottongrass; and various berry-bearing shrubs, *Vaccinium* spp. These species are selected because they are abundant on all the sites, are important links in the trophic structure of the ecosystems of the arctic, and represent a major percentage of the primary production along the coastal plain. The blueberry, huckleberry, and lingonberry, *Vaccinium* spp., are evaluated because of their roles as forage plants and as subsistence species. All of these representative species are evaluated at the Cape Lisburne installation.

**3.2.2.2 Representative Aquatic Invertebrates and Fish.** The potential exposure pathways at Cape Lisburne do not affect any aquatic habitats that are large or deep enough to support fish populations. As a result, no fish species were selected for evaluation in the Cape Lisburne ERA. The invertebrates selected as representative species are *Daphnia* spp. (water fleas). *Daphnia* spp. are abundant (Johnson and Burns 1984; Wootton 1976), and toxicity information is readily available for them. No marine mammals are evaluated in the Cape Lisburne ERA because there are no complete pathways for COCs (at concentrations that are of concern) to reach potential marine receptors.

**3.2.2.3 Representative Birds.** The representative avian species are Lapland longspur; brant, *Branta bernicla*; glaucous gull; and pectoral sandpiper, *Calidris melanotos*. The Lapland longspur is a passerine belonging to a terrestrial feeding guild (including sandpipers, turnstones, and phalaropes) (Custer and Pitelka 1978). The longspur's diet of insects and seeds (Custer and Pitelka 1978) makes it an important link in the arctic trophic web. The brant nests and molts among the numerous ponds in the tussock tundra and grazes on sedges and cottongrass (Palmer 1976). The brant is also considered to be an important subsistence resource. The glaucous gull is a predatory scavenger that feeds on small mammals, young birds, carrion, and garbage, and breeds along the Arctic Coastal Plain (Farrand 1983). The pectoral sandpiper is an abundant shorebird that is primarily insectivorous and breeds on the Arctic Coastal Plain. The Lapland longspur, brant, glaucous gull, and pectoral sandpiper are in potential exposure pathways at the Cape Lisburne installation and will be evaluated in this ERA. All the avian species in this ERA are migratory, and as such, are protected under the Migratory Bird Treaty Act of 1978. This is reflected by the use of a protected species factor of 2 in the calculation of avian toxicity reference values (TRVs).

**TABLE 3-4. REPRESENTATIVE AND SENSITIVE SPECIES AT AIR FORCE ARCTIC RADAR INSTALLATION SITES**

COMMON NAME	GENUS AND SPECIES
<b>PLANTS</b>	
Sedge	<i>Carex</i> spp.
Cottongrass	<i>Eriophorum</i> spp.
Willow	<i>Salix</i> spp.
Berries	<i>Vaccinium</i> spp.
<b>AQUATIC ORGANISMS</b>	
Water fleas	<i>Daphnia</i> spp.
Nine-spined stickleback	<i>Pungitius pungitius</i>
Arctic char	<i>Salvelinus alpinus</i>
<b>BIRDS</b>	
Lapland longspur	<i>Calcarius lapponicus</i>
Brant	<i>Branta bernicla</i>
Glaucous gull	<i>Larus hyperboreus</i>
Pectoral sandpiper	<i>Calidris melanotos</i>
<b>MAMMALS</b>	
Brown lemming	<i>Lemmus trimucronatus</i>
Arctic fox	<i>Alopex lagopus</i>
Barren-ground caribou	<i>Rangifer tarandus</i>
<b>ENDANGERED AND THREATENED SPECIES*</b>	
Spectacled eider <sup>a</sup>	<i>Somateria fischeri</i>
Steller's eider <sup>b</sup>	<i>Polysticta stelleri</i>

\* See Section 3.2.2.5 for information about endangered and threatened species.

a Threatened status.

b Candidate for threatened status, see text for explanation.



**3.2.2.4 Representative Mammals.** The representative species of mammals selected are the brown lemming, arctic fox, and barren-ground caribou. The brown lemming is the predominant small mammal at all installations. The lemming consumes more vegetation than expected for an animal its size, due to its low assimilation efficiency, the low nutrient value of winter forage, and the high metabolic demands of the arctic environment (Chappell 1980). The arctic fox is selected as a representative species because it is ubiquitous along the coastal plain and its carnivorous diet (mostly lemmings) places it near the top of the trophic structure in the arctic. Eberhardt et al. (1982) note that in fall and winter, and to a lesser extent in summer, the arctic fox frequently uses areas near development. This tendency may expose the fox to potential pathways of contamination. Additionally, the fox, a relatively common furbearer, can be an important subsistence resource. The caribou is selected as a representative species because it uses areas on, or near, a number of the radar installations during migration, calving, and post-calving. In addition, the caribou is a significant subsistence resource for local people along the Arctic Coastal Plain (USFWS 1982; Cuccarese et al. 1984; Hensel et al. 1984). The three mammal species discussed may be potentially exposed to COCs at the Cape Lisburne installation and will be evaluated in this ERA.

**3.2.2.5 Threatened and Endangered Species.** The threatened and endangered species that potentially occur at the Air Force arctic radar installations are the spectacled eider and Steller's eider. The spectacled eider is federally listed as threatened, and Steller's eider is a candidate for listing as threatened. The U.S. Fish and Wildlife Service indicated that it was likely that Steller's eider would be listed as threatened sometime in 1995 (Ambrose 1994 pers. comm.), but a federal moratorium on additions to the threatened and endangered lists is in effect. Alaska Biological Research (1994) conducted surveys searching for spectacled and Steller's eiders on and near the Air Force arctic radar installations. The surveys report a low likelihood of either spectacled eiders or Steller's eiders using the habitats at or near the Cape Lisburne installation. The arctic subspecies of peregrine falcon (*Falco peregrinus tundrius*) was delisted by the USFWS, 5 October 1994. As a result, no threatened or endangered species were evaluated in the Cape Lisburne ERA.

### **3.2.3 Exposure Pathways**

This section discusses potential exposure pathways for ecological receptors. In addition, methods used to quantify exposures to selected species of plants, aquatic organisms, birds, and mammals are presented. Quantitative estimates of exposure will be compared with TRVs derived in Section 3.3 to estimate risks in the risk characterization section (Section 3.4).

Ecological receptors can be exposed to COCs through abiotic and biotic media. Potential exposure pathways for terrestrial and aquatic organisms are summarized in Figure 3-1. The following sections describe the potential exposure routes and a determination of pathways evaluated in the risk assessment.

Potential risks to representative species of plants from exposure to COCs in soil and surface water will be addressed. The most significant route of exposure for plants is direct contact with soil at the site, although a qualitative evaluation of the effects of COCs in surface water is presented in Section 3.4.1.

Aquatic organisms such as fish and invertebrates are primarily exposed through direct contact with surface water. Surface water is in direct contact with dermal surfaces as well as gills and other respiratory structures. Fish and invertebrates also may be exposed to COCs through ingestion of plant and animal items in the diet, direct contact with sediments, and incidental ingestion of sediments while foraging. Direct contact with surface water is the primary exposure route, however, and these secondary routes (ingestion) will not be evaluated for aquatic organisms.

Wildlife, such as birds and mammals, may be exposed to COCs through a variety of pathways including ingestion of surface water used for drinking, ingestion of plant and animal diet items, and incidental ingestion of surface soils and sediments while foraging. Wildlife species are not expected to be exposed to COCs via inhalation because the surface soils are well vegetated and moist during the growing season and frozen and/or snow covered the remainder of the year. Therefore, the inhalation pathway is not evaluated in the ERA.

Insufficient toxicity and exposure information is available for the representative species to allow quantification of exposures from dermal contact with soil or sediments; therefore, these pathways are not quantitatively evaluated. Because soils and sediments represent potential pathways, total exposures for the representative species could be underestimated. This represents one of the uncertainties in this risk assessment that are discussed in Section 3.5.

The soil and sediment samples at the Cape Lisburne installation were taken at depths ranging from the surface to six feet deep. Only those samples at or above 1.5 feet deep were used in the ERA because the potential exposure pathways for the representative species are likely to be incomplete at depths greater than 1.5 feet. It is unlikely that any of the representative species would be exposed to any soil or sediments much below one foot, although the brown lemming is reported to burrow up to one foot deep (Nowak 1991). As a result, samples below 1.5 feet are not considered ecologically relevant for the representative species being evaluated. This depth-oriented sample screening contributes to a conservative risk assessment (a potential overestimation of risk).

#### **3.2.4 Habitat Suitability for Representative Species**

The habitat suitability evaluation considered the representative aquatic, avian, and mammalian species selected for evaluation at the Cape Lisburne installation.

Human development and activities at the Cape Lisburne installation have impacted the natural habitats available to the representative species. The effects of these impacts are uncertain; in some cases the activities probably deter wildlife use of the area and in other cases they may attract wildlife (e.g., arctic fox and gulls attracted to a landfill). Although these impacts may affect how and when representative species may use the habitats at Cape Lisburne, the impacts are not subject to quantification, and as a result, all the sites at the Cape Lisburne installation are evaluated in this ERA. In some cases, the media-specific samples have been taken at locations that do not represent suitable habitat for all the representative species (e.g., under-building sample locations that are obviously not suitable for caribou, or surface water sample locations that are not accessible to fish species). This may result in an overestimate of exposure because

sample data from all locations are used to calculate the average concentrations which, in turn, are used to estimate exposure for the representative species. These conditions will be noted in the risk characterization and uncertainty sections presented in the ERA (Sections 3.4 and 3.5, respectively).

The ERA is being conducted for the entire Cape Lisburne installation, but only a portion of the facility consists of potentially contaminated sites. The sites are estimated to total approximately 11.1 hectares (ha), or 27.7 acres. The areal extent of the potentially contaminated sites has been estimated using site maps. The spatial extent of the sites is considered when estimating the onsite dietary intake (IS) in Section 3.2.7.2. In general, based on professional judgement and onsite observations but not on site-specific surveys, the installation provides habitat less suitable than nearby areas because of the numerous roads, gravel pads, human activities, and overall development.

### **3.2.5 Exposure Assessment for Representative Species of Plants**

The harsh environment of the Arctic Coastal Plain imposes many restrictions on plant life. The presence of permafrost limits infiltration and percolation of water, so the water table is often at or above the surface. The vast majority of plant species is perennial, with much of their biomass (50 to 98 percent) underground (Raven et al. 1986). The potential pathways of contamination for plants are through the soil/sediment and surface water.

*Carex* spp., *Salix* spp., and *Eriophorum* spp. all store food reserves in rhizomes. Mycorrhizal fungi play an important role in the transport and delivery of nutrients to the rhizomes and the roots of these species. This underground system probably developed in response to the harsh aboveground arctic environment. As a result, surface water contaminated with chemicals that are lighter than water (i.e., petroleum and its derivatives) does not present a greatly increased hazard to the below-ground portion of plants. This has been shown experimentally by exposing arctic coast vegetation to petroleum products (Walker et al. 1978). The experiments showed that sedges, willows, and cottongrass plants were not adversely affected by low to moderate amounts of petroleum (spill concentrations in the studies were up to 12 L/m<sup>2</sup>) in wet environments. Thus, soil/sediment will be considered the primary pathway of potential contamination for plants. The chemical concentration used in the Risk Characterization (Section 3.4) is the average concentration of the COC in the soil/sediments at the installation. A qualitative evaluation of the effect of potentially contaminated surface water on plants is presented in Section 3.4.1.

### **3.2.6 Exposure Assessment for Representative Aquatic Organisms**

Organisms that dwell in an aquatic environment are exposed to chemicals contained in the water column. For this reason, the exposure assessment considers the concentrations in surface water to be the exposure concentrations to aquatic organisms. As described in Section 3.2.3, the primary exposure route for aquatic organisms is direct contact with surface water, and as a result, the aquatic representative species are not evaluated for contact with, or ingestion of, sediments. The risk assessment compares the average concentration of the COCs found in surface waters to toxicity data for the representative aquatic species to calculate the risk estimate.

### 3.2.7 Exposure Estimates for Representative Bird and Mammal Species

Exposure estimates for the representative species of mammals and birds (expressed as a unit of chemical ingested per unit of body weight) are based on their total exposure to COCs from diet, soils, and surface water using the following equation:

$$EE = [(FI \times CF) + (WI \times CW) + (SI \times CS \times ROA)] \times UCF \times IS / BW$$

where:

EE	=	estimated exposure (mg/kg-bw/day).
FI	=	food intake rate (g/day); rates are derived in the life history tables. Diets (both vegetable and animal components) are proportioned according to the diet composition information in the life history tables and are presented below.
CF	=	chemical concentration in food (mg/kg); based on concentrations for each group of food items.
WI	=	water intake rate (L/day); rates are derived in the life history tables.
CW	=	chemical concentration in water (µg/L); see Section 3.1 for calculations of concentrations.
SI	=	soil/sediment intake rate (g/day); based on a percentage of food intake.
CS	=	chemical concentration in soil/sediment (mg/kg); see Section 3.1 for calculations of concentrations.
ROA	=	relative oral availability; default to 1.0 (lack of information). This value assumes that the bioavailability of the chemical in the test medium is the same as for the medium onsite.
UCF	=	0.001; unit conversion factor used in conversion of g to kg, µg to mg, and L to ml, to ensure EE is reported in mg/kg-bw/day.
IS	=	fraction of dietary intake at potentially contaminated sites (by weight).
BW	=	body weight (kg).

In the case of species that have partial herbivorous dietary intakes, the CF x FI phrase in the equation is multiplied by the proportion of vegetation in their diet (these calculations are presented in Appendix D, Concentration in Food Calculations). Those species and their respective proportions are: Lapland longspur, 0.25; brant, 0.90; glaucous gull, 0.10; and pectoral sandpiper, 0.10 (see the life history tables, Tables 3-6 to 3-9, for references regarding the proportion of vegetation in the species' diets). The estimated exposure calculations for bird and mammal receptors are presented in Appendix E.

**3.2.7.1 Potential Bioaccumulation of COCs in Representative Species.** The potential risks from ingestion of COCs in dietary items are difficult to determine because of the complexity of the trophic web. Inputs to the exposure estimate equation include concentrations of contaminants in water and soil, ingestion rates for water, food, and soil, the relative use of the potentially contaminated sites compared to the representative species' normal range, and body weight. The food ingested, in the case of higher level consumers, may be from different levels of the trophic web. For example, a contaminant may be taken up by a plant, which is consumed by a lemming, which is then eaten by an arctic fox. The amount of contaminant to which the fox

is exposed is not readily quantified without supporting empirical data at each trophic level. Because of this and the lack of data to assist in quantifying bioaccumulation, the risk assessment does not account for bioaccumulation in the animal portion of the trophic web. This uncertainty is tempered by the "hot spot" nature of the distribution of the COCs. It is possible that representative species may be exposed to these "hot spots" occasionally, but it is unlikely that their entire exposure will occur at these locations. Use of the average concentrations may overestimate the potential exposure of representative species (this is discussed in more detail in the ERA Uncertainty Analysis, Section 3.5.1). Furthermore, the likelihood of predators repeatedly taking prey that have been exposed to a COC "hot spot" is low. For example, the arctic fox ranges over such a wide area that any COCs to which the fox would be exposed via bioaccumulation would represent only a very small proportion of its overall exposure.

Further, most of the COCs in soils/sediments at the Cape Lisburne installation were petroleum hydrocarbons, BTEX, and VOCs, which are unlikely to bioaccumulate. PCBs, also COCs in soil/sediment, are known to bioaccumulate, and this is addressed further in Section 3.4.5, Potential Future Risks. For illustrative purposes, bioconcentration factors (BCF) calculated (Veith et al. 1979 in Spacie and Hamelink 1985) for some organic COCs are presented in Table 3-5.

Based on these low BCFs, the exposure estimates for organic chemicals do not include potential bioaccumulation of COCs in the animal portion of the trophic web. It is unlikely that the organic chemicals will bioaccumulate (based on the concentrations reported in the soil and water), such that the exposure estimates would exceed, or even approach, the TRVs. BCF calculations are presented in Appendix C.

The inorganic COCs at Cape Lisburne, iron and manganese, were detected in surface water. Surface water concentrations are not used to quantify potential plant uptake, but the dissolved iron and manganese concentrations (810 and 337  $\mu\text{g/L}$ , respectively) at Cape Lisburne are within the range of background concentrations for these COCs. The bioaccumulation of metals in the animal portion of the trophic web is not amenable to quantification without sample concentrations at each level of consumer. Information about the bioaccumulation of iron and manganese is not available in the literature, but because these COCs are essential nutrients, it is likely that the metabolic processes that make use of iron and manganese will prevent undue bioaccumulation.

**3.2.7.2 Estimation of Percent Ingested Onsite.** The size of the areas used by the representative species, and hence their potential exposure to COCs, varies greatly. Generally, a species' home range is used to characterize the size of the area it uses on a regular basis (disregarding migration and dispersal). This information, combined with the extent of the potentially contaminated sites, can be used to estimate the percent of dietary intake that a species gets from the sites.

This estimate is referred to as the "percent of dietary intake at sites" (IS) value in the exposure estimate equation. The IS value is represented by the ratio of the total area of the sites (11.1 ha or 27.7 acres) to the reported home range size (or converted population density values) for the representative species. When home range information for a species was not available, population density values have been converted to estimate the area used by the species. The representative species are most likely at Cape Lisburne during, or directly after, the breeding

**TABLE 3-5. BIOCONCENTRATION FACTORS FOR SELECTED ORGANIC COMPOUNDS IN WATER**

CHEMICAL	Log K <sub>ow</sub>	BCF
DRPH	5.30	6,238
Naphthalene	3.36	211
Trimethylbenzene	3.78	439
Xylene	3.16	149

Note: BCF calculated from Log K<sub>ow</sub> according to the following equation:  
 $\text{Log BCF} = 0.76 \text{ Log K}_{ow} - 0.23$  (Veith et al. 1979 in Spacie and Hamelink 1985).  
K<sub>ow</sub> = octanol/water partition coefficient.

season, when many species become territorial. These territories represent the area used by the species. The densities of the population may provide estimates of the size of the territories and are used as substitute values when home range information is unavailable. This presents an added degree of uncertainty (see Section 3.5.3). If the home range (or converted population density value) is less than the total areal extent of the sites (11.1 ha), the maximum value for IS is 1.0 because it is possible that a species could meet all its dietary intake needs within the potentially contaminated areas. The IS values for the representative bird and mammal species are given below. Note that this is a conservative estimate because the 11.1 ha size assumes that the contaminated sites are the only area used. Obviously, the species would use the suitable areas between the potentially contaminated sites, and this results in potential exposure to COCs less than if the species restricted its location to contaminated sites only.

**Birds.** Lapland longspur. IS = 1.0; Derksen et al. (1981) report a breeding density of 38.6 birds/km<sup>2</sup>. This corresponds to about 1 bird/2.6 ha, and results in a ratio of 11.1:2.6, or 4.3. The IS value defaults to the maximum of 1.0.

Brant. IS = 0.56; density of breeding pairs reported by Derksen et al. (1981) is 5.0 birds/km<sup>2</sup>. At this density of 1 brant/20 ha, the total extent of the potentially contaminated sites is about 56 percent of the area a brant might use.

Glaucous gull. IS = 0.09; the density for the glaucous gull is reported by Derksen et al. (1981) as 0.8 birds/km<sup>2</sup>. This density, about 1 gull/125 ha, yields an IS value of approximately 0.09.

Pectoral sandpiper. IS = 1.0; the density of the pectoral sandpiper along the Arctic Coastal Plain is reported by Derksen et al. (1981) as 22.4 birds/km<sup>2</sup>. This density equates to one sandpiper/4.5 ha, and the corresponding IS value is the maximum of 1.0.

**Mammals. Brown lemming.** IS = 1.0; the lemming's home range is reported as 0.5 ha (Nowak 1991), resulting in the possibility that several lemmings may consume all their dietary needs within the bounds of a site. The lemming is not likely, however, to use the wetter sites (which constitute a large portion of the total extent of the sites) where the majority of the contaminant pathways are located. Also, the potentially contaminated sites are mostly gravel pads that have been constructed for development purposes, support little or no vegetation, and offer a poor matrix for the lemming to use for burrowing. For these reasons the IS used for the brown lemming is 0.5 rather than 1.0.

**Arctic fox.** IS = 0.03 ; the home range of the fox is extremely variable. Eberhardt et al. (1982) report a home range of 3.7 to 20.8 km<sup>2</sup> for juvenile and adult arctic foxes, respectively. Using the juveniles' home range estimate (3.7 km<sup>2</sup> or 370 ha), the IS value for the arctic fox is 0.03.

**Caribou.** IS = 0.01; caribou are highly mobile, covering large distances during their movements to and from calving grounds and in their constant search for suitable forage. They may range over thousands of kilometers a year, and as a result there is no accurate estimate of their home range. Based on professional judgement and knowledge of the caribou's habits, a very conservative estimate of the likelihood of caribou using the potentially contaminated sites is 1 percent, or the corresponding minimum IS value of 0.01.

**3.2.7.3 Exposure Assessment for Representative Species of Birds.** In this section the methods for quantifying exposures to the selected representative species of birds are presented.

In order to estimate exposures of the representative species of birds, life history information was compiled for the selected species. This information includes: occurrence at the Air Force arctic radar sites, habitat, average body weight, estimated food intake rate, estimated water intake rate, diet composition, and home range and/or population density. The life history information is presented at the end of Section 3.2 in Tables 3-6 through 3-9 and 3-11 through 3-13.

Plant uptake of contaminants has been quantified for use in the exposure estimations for herbivores (bird and mammal species). Herbivores are potentially exposed to contamination directly from ingestion of soil and water intake as well as through their diet. The dietary plant component (CF in the exposure estimate equation) is calculated by multiplying the contaminant's soil concentration by the BCF,  $B_v$ .  $B_v$  is defined as the ratio of the concentration in aboveground parts of a plant (mg of compound/kg of dry plant) to the concentration in soil (mg of compound/kg of dry soil). The  $B_v$  can be used to predict the level of a potential contaminant taken up by a plant, and this information can then be used to assess the potential transport of the contaminant in the trophic web.

The uptake of metals by plants is quantified using the  $B_v$  values in Baes et al. (1984). These values represent potential uptake to the vegetative portions of the plant. The approach for organic chemicals is basically the same, except that the  $B_v$ s for organic chemicals are derived using a regression equation (Travis and Arms 1988). The equation is:

$$\log B_v = 1.588 - 0.578 (\log K_{ow})$$

**TABLE 3-6. LIFE HISTORY INFORMATION FOR THE LAPLAND LONGSPUR, *Calcarius lapponicus***

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Seasonal breeder at coastal arctic radar stations	Dominant breeding passerine	U.S. Air Force 1993
Habitat	Breeds on arctic coastal tundra		Scott 1983
Body Weight	27.3 g (0.027 kg)	Mean of 68 specimens	Dunning 1984
Food Intake Rate	6.5 g/day dry matter	FI = 0.141 (BWkg) <sup>0.850</sup>	Nagy 1987
Water Intake Rate	0.005 liters/day	WI = 0.059 (BWkg) <sup>0.67</sup>	Calder and Braun 1983
Diet Composition	During breeding (June and July): insects (crane flies); pre- and post-breeding (May and August): seeds (grasses); average 25 percent vegetation in diet	Passerine member of insectivorous foraging guild which includes shorebirds	Custer and Pitelka 1978
Population Density	38.6/km <sup>2</sup>	Varies with changing predation pressures	Derksen et al. 1981

**TABLE 3-7. LIFE HISTORY INFORMATION FOR THE BRANT, *Branta bernicla***

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Seasonal breeder at or near coastal arctic radar stations	Breeding, migratory sp., subsistence sp.	U.S. Air Force 1993
Habitat	Breeds on Arctic Coastal Plain	Prefers low, barren, wet, coastal terrain	Palmer 1976
Body Weight	1,305 g (1.305 kg)	Mean of 791 specimens	Dunning 1984
Food Intake Rate	69.2 g/day dry matter	FI = 0.0582 (BWkg) <sup>0.651</sup>	Nagy 1987
Water Intake Rate	0.07 liters/day	WI = 0.059 (BWkg) <sup>0.67</sup>	Calder and Braun 1983
Diet Composition	Sedges, grasses; average 90 percent vegetation in diet	Some insects during breeding (June and July)	Palmer 1976
Population Density	5.0/km <sup>2</sup>	Average from three coastal sites	Derksen et al. 1981



**TABLE 3-8. LIFE HISTORY INFORMATION FOR THE GLAUCOUS GULL *Larus hyperboreus***

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Seasonal breeder and migrant at coastal arctic radar stations	Relatively common along arctic coast	Woodward-Clyde 1993
Habitat	Coastal tundra, lakes, ponds, and marine environment	Breeds on arctic coast	Farrand 1983
Body Weight	1,445 g (1.445 kg)	Mean of 65 specimens	Dunning 1984
Food Intake Rate	74 g/day dry matter	FI = 0.0582 (BWkg) <sup>0.651</sup>	Nagy 1987
Water Intake Rate	0.08 liters/day	WI = 0.059 (BWkg) <sup>0.67</sup>	Calder and Braun 1983
Diet Composition	Small fish, birds, insects, crustaceans, mollusks, and garbage; average 10 percent of vegetation in diet	Predatory scavenger	Martin et al. 1961
Population Density	0.8/km <sup>2</sup>	Average from three coastal sites	Derksen et al. 1981

**TABLE 3-9. LIFE HISTORY INFORMATION FOR THE PECTORAL SANDPIPER *Calidris melanotos***

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Seasonal breeder at coastal arctic radar stations	Abundant on Arctic Coastal Plain	Woodward-Clyde 1993
Habitat	Grassy margins of wet meadows, marshes, riparian areas, and ponds	Nests hidden on well-drained grassy sites	Scott 1983; Martin et al. 1961
Body Weight	79 g (0.079 kg)	Mean of 35 specimens	Dunning 1984
Food Intake Rate	11.2 g/day dry matter	FI = 0.0582 (BWkg) <sup>0.651</sup>	Nagy 1987
Water Intake Rate	0.01 liter/day	WI = 0.059 (BWkg) <sup>0.67</sup>	Calder and Braun 1983
Diet Composition	Insects, mollusks, crustaceans, worms, and vegetable debris; average 10 percent vegetation in diet	Craneflies are major diet component	Martin et al. 1961; Pitelka 1959
Population Density	22.4/km <sup>2</sup>	Average from three coastal sites	Derksen et al. 1981

**TABLE 3-10. SOIL INGESTION ESTIMATES FOR REPRESENTATIVE BIRD SPECIES**

REPRESENTATIVE SPECIES	SURROGATE SPECIES	ESTIMATED PERCENT OF SOIL IN DIET	ESTIMATED g/day SOIL IN DIET
Lapland longspur	No suitable surrogate	<2.0	0.1
Brant	Canada goose	8.2	5.7
Glaucous gull <sup>a</sup>	Siberian glaucous gull	7.6	5.6
Pectoral sandpiper	Four sandpiper species (average)	18.1	2.0

<sup>a</sup> Information from Belopol'skii 1961.  
Source: Beyer et al. 1994

**TABLE 3-11. LIFE HISTORY INFORMATION FOR THE BROWN LEMMING *Lemmus trimucronatus***

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Resident at coastal arctic radar stations	Dominant small mammal	U.S. Air Force 1993
Habitat	Tundra and alpine meadows	Nests above ground in winter, below in summer	Burt and Grossenheider 1976
Body Weight	55 g (0.055 kg)		Chappell 1980
Food Intake Rate (dry matter)	24-45 g/day	Has low assimilation efficiencies (31 to 36 percent), variation also related to seasons	Chappell 1980
Water Intake Rate	0.007 liters/day	$WI = 0.099 (BWkg)^{0.90}$	Calder and Braun 1983
Diet Composition	Sedges, grasses, lichens, roots, leaves, bark, berries		Nowak 1991
Home Range Size (AVG)	0.5 ha (females) 1.0 ha (males)	0.5 ha used in assessment	Nowak 1991
Population Density	0 to 325/ha	Populations have large fluctuations on a three to five year cycle; currently populations are low	Nowak 1991; Snyder-Conn 1994

**TABLE 3-12. LIFE HISTORY INFORMATION FOR THE ARCTIC FOX, *Alopex lagopus***

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Resident at coastal arctic radar stations	Ubiquitous	U.S. Air Force 1993
Habitat	Tundra and coastal plain	Dens in sandy mounds >1 m high	Chesemore 1967
Body Weight	4,950 g (4.95 kg)		Burt and Grossenheider 1976
Food Intake Rate	256 g/day dry matter	FI = 0.0687 (BWkg) <sup>0.822</sup>	Nagy 1987
Water Intake Rate	0.42 liters/day	WI = 0.099 (BWkg) <sup>0.90</sup>	Calder and Braun 1983
Diet Composition	Brown lemming (summer), nesting birds, carrion, seal pups, non-food items	Brown lemming in >85 percent of all scats, n = 224	Chesemore 1967; Nowak 1991
Home Range Size (AVG)	20.8 km <sup>2</sup> adult 3.7 km <sup>2</sup> juvenile (<1 yr)	Adult range used in assessment	Eberhardt et al. 1982

**TABLE 3-13. LIFE HISTORY INFORMATION FOR THE BARREN-GROUND CARIBOU**  
*Rangifer tarandus*

PARAMETER	VALUE	NOTES	REFERENCE
Occurrence at Arctic Radar Sites	Seasonal, at or near coastal arctic radar stations during migrations	Some sites used for calving	U.S. Air Force 1993
Habitat	Tundra in summer, open coniferous forest in winter	Varies, related to migration	Burt and Grossenheider 1976
Body Weight	95,500 g (95.5 kg)	Mean for adults, male, and female	Nowak 1991
Food Intake Rate	2,400 g/day (2.4 kg) dry matter	FI = 0.0875 (BWkg) <sup>0.727</sup>	Nagy 1987
Water Intake Rate	6.0 liters/day	WI = 0.099 (BWkg) <sup>0.90</sup>	Calder and Braun 1983
Diet Composition	Willows, sedges, cottongrass, lichens	Selection based on plant phenology	Skogland 1980; White and Trudell 1980
Population Density	1.41 km <sup>2</sup>	Undisturbed calving area	Cameron et al. 1992
	0.31 km <sup>2</sup>	Within 1 km of road	
	4.53 km <sup>2</sup>	Within 5-6 km of road	

where:

$B_v$  = the BCF (unitless) and  
 $K_{ow}$  = the octanol-water partition coefficient of the chemical (mol/m<sup>3</sup> / mol/m<sup>3</sup>)

In order to calculate the potential uptake of DRPH by plants, the  $K_{ow}$  of diesel fuel was estimated. The estimation of the  $K_{ow}$  was conducted using equation 2-3 in Lyman et al. (1981):

$$\log S = -0.922 \log K_{ow} + 4.184$$

where:

$S$  = solubility (mg/L) and  
 $K_{ow}$  = octanol/water partition coefficient (mol/m<sup>3</sup> / mol/m<sup>3</sup>)

This equation estimates the solubility of an organic chemical in water. However, it may also be manipulated arithmetically to calculate the log  $K_{ow}$  based on the known solubility:

$$\log K_{ow} = \frac{\log S - 4.184}{-0.922}$$

The solubility of diesel fuel (0.2 mg/L) (Custance et al. 1992) was used to calculate the log  $K_{ow}$  of diesel fuel. The log  $K_{ow}$  is calculated to be 5.3.

Life history information for the Lapland longspur, brant, glaucous gull, and pectoral sandpiper is presented in Tables 3-6 through 3-9.

Information is not available on the daily food intake rate (grams/day) and water intake rate (liters/day) for the representative bird species in the arctic habitat. Therefore, this information was estimated using regression equations associated with body weight (Calder and Braun 1983). The severity of the arctic climate may impose higher metabolic demands on animals. As a result, the food and water intake rates should be considered estimates only, and their uncertainty should be kept in mind. The food intake rate was estimated using Nagy's (1987) equations:

Passerine birds (i.e., Lapland longspur):

$$FI \text{ (kg/day dry matter)} = 0.141 \times (\text{body weight in kilograms})^{0.850}$$

All other birds:

$$FI \text{ (kg/day dry matter)} = 0.0582 \times (\text{body weight in kilograms})^{0.651}$$

The water intake rate was estimated using the equation developed by Calder and Braun (1983):

All birds:

$$WI \text{ (liters/day)} = 0.059 \times (\text{body weight in kilograms})^{0.67}$$

As animals forage they may incidentally ingest soil and sediment particles. The average concentration of contaminants in soil/sediment can be multiplied by the amount of soil/sediment ingested to estimate the potential uptake of contaminants by this route. Soil intake rates have been reported for just a few wildlife species (Beyer et al. 1994). The soil ingestion rates for the representative species are extrapolated from Beyer et al. (1994) by using similar species with reported values. The percentages reported are of the total weight of dietary intake. Table 3-10 lists the representative bird species, the species used as surrogates, and the estimated percentages of soil ingested in quantifying exposure to contaminants. Species that forage directly in the soil or sediment, such as the sandpiper or brant, show relatively high percentages of soil in their diet. The Lapland longspur does not have appropriate surrogate species with soil ingestion data. Although the longspur is in the same foraging guild as sandpipers (which incidentally ingest relatively large amounts of soil), the longspur takes insects from the soil surface or gleans its prey from vegetation (Custer and Pitelka 1978), thus minimizing its soil intake. Its estimate of soil ingestion (less than 2 percent of diet by weight) reflects this. The glaucous gull ingests stones and sand as a mechanical addition (to aid in digestion) to its diet (Belopol'skii 1961), and this contributes to its soil/sediment intake. For those species without a suitable surrogate (i.e., Lapland longspur) or whose soil ingestion rate is reported as <2 percent, a value of 2 percent of dietary intake (by weight) was used to calculate the exposure estimates.

**3.2.7.4 Exposure Assessment for Representative Species of Mammals.** This section assesses exposure to contaminants for the selected representative species of mammals. Table 3-11 (brown lemming), Table 3-12 (arctic fox), and Table 3-13 (caribou) present life history data are used to calculate exposure estimates for the representative species. Home range and/or population density has been listed for the representative mammal species, depending on appropriateness and availability.

Information on daily food intake rates for the arctic fox and caribou was not available. The rates have been estimated using regression equations associated with average body weights and metabolic rates (Nagy 1987). The food intake rates for the fox and caribou were estimated using the following equations, developed for placental mammals in general and for herbivorous mammals, respectively (Nagy 1987).

arctic fox: using equation for placental mammals in general

$$FI \text{ (kg/day dry matter)} = 0.0687 \times (\text{body weight in kilograms})^{0.822}$$

caribou: using equation for mammalian herbivores

$$FI \text{ (kg/day dry matter)} = 0.0875 \times (\text{body weight in kilograms})^{0.727}$$

Because of very low assimilation efficiencies, the low nutrient content of winter forage, and the high metabolic demands in arctic habitats (Chappell 1980), the equation for food intake rate significantly underestimates the rate for the brown lemming. A more appropriate rate for the brown lemming of 45 g/day is reported by Chappell (1980) (using the highest value in the reported range of 24 to 45 g/day).

The rates for water intake of the representative mammals were estimated using the equation generated by Calder and Braun (1983) because of the unavailability of species-specific information in the literature. The equation is:

$$WI \text{ (liters/day)} = 0.099 \times (\text{body weight in kilograms})^{0.90}$$

Incidental soil intake was evaluated for mammals in the same manner as for birds (Section 3.2.7.3). Table 3-14 shows the percent of soil ingested by the representative mammal species.

### **3.3 ECOLOGICAL TOXICITY ASSESSMENT**

This section presents toxicity information for each COC in surface water and soils/sediments. The COCs identified in surface water are iron, manganese, and 1,2,4-trimethylbenzene. The COCs in soils/sediments are DRPH, RRPH, GRPH, benzene, toluene, xylenes, 1,4-dichlorobenzene, 4-methylphenol, Aroclor 1254, and Aroclor 1260. Sections 3.3.1 through 3.3.10 discuss the toxicity of all COCs to the receptor groups. Section 3.3.10 presents the derivation of TRVs used for this ERA.

#### **3.3.1 Petroleum Hydrocarbons**

Section 3.1 presented the COCs for sites at the Cape Lisburne installation. DRPH, GRPH, and RRPH were identified as COCs in soils/sediments. This section is a discussion of the chemical differences between DRPH, GRPH, and RRPH and the toxicity of these three petroleum mixtures.

Crude petroleum contains thousands of different chemical compounds. Gasoline and diesel fuel are refined petroleum products. The composition of gasoline and diesel fuel depends not only on the origin of the crude oil from which the gasoline is derived, but also the process technique

**TABLE 3-14. SOIL INGESTION ESTIMATES FOR REPRESENTATIVE MAMMAL SPECIES**

REPRESENTATIVE SPECIES	SURROGATE SPECIES <sup>a</sup>	ESTIMATED PERCENT OF SOIL IN DIET <sup>a</sup>	ESTIMATED g/day SOIL IN DIET
Brown lemming	Prairie dog (average of two species)	6.2	1.2
Arctic fox	Red fox	2.8	7.2
Caribou	Elk	<2.0	48

<sup>a</sup> Beyer et al. 1994.

and the blending scheme (Von Burg 1993). Once gasoline or diesel fuel is released to the environment, weathering and volatilization further alter its composition.

Gasoline is a complex, highly variable mixture of petroleum hydrocarbons containing 3 to 21 carbon atoms; however, compounds with 4 to 12 carbon atoms predominate. Gasoline is detected with the petroleum hydrocarbon analysis as GRPH. The following chemical classes are detected as GRPH: paraffins (straight-chained alkanes), olefins (straight-chained alkenes), naphthenes (cycloalkanes and alkenes), and aromatic hydrocarbons (alkylbenzenes and polynuclear) (Von Burg 1993). Although GRPH are generally in the range of 4 to 12 carbon atoms, the laboratory that conducted the analyses for Cape Lisburne detected GRPH with 6 to 9 carbon atoms. As many as 140 compounds have been identified as constituents of gasoline; however, a small number of constituents such as benzene drive the toxicity. Diesel fuel is also a complex, variable mixture of the same classes of compounds containing 6 to 21 carbon atoms. Diesel fuel is detected with petroleum hydrocarbon analysis as DRPH. The laboratory that analyzed samples for Cape Lisburne detected DRPH with 10 to 24 carbons atoms. As many as 45 compounds have been identified as constituents of diesel fuel (Von Burg 1993). DRPH could include many different types of chemicals, although the majority of molecules would include 24 carbon atoms or more.

Table 3-15 presents the chemical classes and weight percent for GRPH and DRPH. Generally, gasoline contains more aromatic compounds and simple-chained alkanes, whereas diesel fuel is characterized by cycloparaffins (or cycloalkanes). Both gasoline and diesel fuel will be affected by the environment. Weathering will change the chemical composition of petroleum, and specifically, concentrations of aromatic compounds such as benzene will decrease as a result of volatilization. Available toxicity test data have been derived from pure, fresh product, and therefore the applicability to the weathered product encountered at Cape Lisburne is uncertain. Gasoline is the most studied of the petroleum products; however, most data are based on inhalation studies. Gasoline was classified by EPA (1992d) as a Group C (possible human) carcinogen, whereas diesel oil was classified as Group D (not classifiable as to human carcinogenicity). Presumably, this classification of gasoline is due to benzene which, under the conditions of environmental exposure, would volatilize more rapidly than any other constituent.

**TABLE 3-15. CHEMICAL CLASSES OF GRPH AND DRPH**

CHEMICAL CLASS	WEIGHT PERCENT (%) <sup>a</sup>
<b>GRPH<sup>b</sup></b>	
Normal paraffins (n-alkanes)	19.3-38.4 (28.8)
Isoparaffins (isoalkanes)	11.5-50.3 (30.9)
Naphthenes (cycloparaffins or cycloalkanes)	1.0-2.8 (1.9)
Aromatics (e.g., benzene, toluene, pyrene)	9.7-54.7 (32.2)
<b>DRPH<sup>c</sup></b>	
Normal paraffins (n-alkanes)	5.6
Isoparaffins (isoalkanes)	11.1
Naphthenes (cycloparaffins or cycloalkanes)	46.3
Aromatics (e.g., benzene, toluene, pyrene)	33.3
Nitrogen, sulfur and oxygen compounds	3.7

<sup>a</sup> Average shown in parentheses.

<sup>b</sup> Heath et al. 1993.

<sup>c</sup> Weeks et al. 1988.

The gasoline and diesel petroleum hydrocarbon data from surface water samples collected at Cape Lisburne indicate that benzene was not detected in surface water, and the soil/sediment samples showed benzene concentrations above action levels. Physical-chemical data from the literature indicates that total petroleum hydrocarbon (TPH) in soil would reflect all constituents with eventual loss of aromatic (e.g., BTEX) components first, lighter alkanes second, lighter polynuclear aromatic hydrocarbons (PAHs) third, followed by naphthalenes. For an old diesel or petroleum spill, TPH measurements may reflect predominantly trace amounts of high molecular-weight PAHs or higher molecular-weight and branched alkanes [Massachusetts Department of Environmental Protection (MDEP) 1993].

For the purposes of ranking the toxicity of GRPH, DRPH, and RRP, it is assumed that BTEX and lighter-weight alkanes were significantly weathered from exposure to the arctic environment, and



that toxicity is more dependent upon noncarcinogenic endpoints associated with alkanes, alkenes, and cycloalkanes. In addition, the toxicity of DRPH and RRPH is associated with the PAH content. However, at Cape Lisburne, only two PAHs were detected: naphthalene and 2-methylnaphthalene, chemicals considered to be noncarcinogenic. Carcinogenic PAHs such as benzo(a)pyrene were not detected. MDEP (1993) reviewed the noncarcinogenic toxicological endpoints in laboratory animals for diesel fuel and gasoline and determined that diesel fuel was an order of magnitude more toxic than gasoline, although other sources indicate that the toxicity of alkanes and cycloalkanes is similar (Armstrong Laboratory 1994; Sax and Lewis 1989). A review of the Cape Lisburne data indicates that DRPH are present at higher concentrations than GRPH and in a range similar to RRPH. Specifically, average concentrations of DRPH were over 19 times higher than average concentrations of GRPH and just less than twice (1.9 X) the average RRPH concentration. As a result, based on the MDEP review and the chemical data reported for the Cape Lisburne surface water and soil/sediment samples, DRPH are evaluated and used to conservatively represent ecological risks from petroleum hydrocarbon contamination (i.e., GRPH, RRPH, and other constituent chemicals, such as 2-methylnaphthalene).

As discussed above, diesel fuel is comprised of a complex mixture of paraffins (straight-chained alkanes), olefins (straight-chained alkenes), naphthenes (cycloalkanes and alkenes), and aromatic (alkylbenzenes, and polynuclear) petroleum hydrocarbons containing 6 to 21 carbon atoms. Hydrocarbons containing 8 to 18 carbon atoms predominate (Von Burg 1993). There are six grades of diesel fuel (Diesel Oil No. 1, Diesel Oil No. 2, Diesel Oil No. 4, Fuel Oil No. 1, Fuel Oil No. 2, and Home Heating Oil) (Von Burg 1993). The specific components of diesel are expected to change from source to source, so the toxicity of diesel fuels is expected to be variable. The following sections summarize the toxicity of diesel fuel to plants, aquatic organisms, birds, and mammals.

**3.3.1.1 Plants.** Petroleum released to the aquatic environment is expected to be toxic to aquatic plants. Toxicity tests have shown that the water-soluble components of petroleum are toxic to an algal species (*Chlorella vulgaris*) (Kauss and Hutchinson 1975). However, in this specific study, the toxicity was short term. The algal community recovered after a "lag phase". It was theorized (Kauss and Hutchinson 1975) that this trend was due to the loss of highly volatile fractions from the testing chamber over time. Exposure to water extracts of No. 2 Fuel Oil depressed algal biomass in communities and resulted in blue-green algal dominance and decreased diatom occurrence (Bott and Rogenmuser 1978).

**3.3.1.2 Aquatic Organisms.** Studies have shown that freshwater arctic zooplankton may be more sensitive to oil pollution than any other arctic freshwater organisms (O'Brien 1978). Geiger and Buikema (1981 in Hedtke and Puglisi 1982) estimated an LC<sub>20</sub> (concentration lethal to 20 percent of the test organisms) of No. 2 Fuel oil to *Daphnia pulex* of 5,600 µg/L (concentration used to calculate TRV).

**3.3.1.3 Birds.** Petroleum hydrocarbons in the environment may affect bird reproduction. External application of No. 2 Fuel Oil to mallard (*Anas platyrhynchos*) and common eider (*Somateria mollissima*) eggs significantly increased embryo mortality (Albers 1977; Szaro and Albers 1977). Mallard eggs were treated with 1, 5, 10, 20, and 50 µl of fuel oil. Ingestion of crude oil by mallards at a concentration of five percent by weight in the diet resulted in

depressed growth (Szaro et al. 1978). Hartung (1964) demonstrated a decrease in weight gain in mallards during the first 10 days after receiving 6,000 mg/kg No. 2 Fuel Oil (concentration used to calculate TRV). However, after 34 days there was no difference between treatment groups and the controls.

**3.3.1.4 Mammals.** The available literature does not present a great deal of information regarding the toxicity of diesel fuel to mammals. The toxicity of diesel fuel to mammals can be represented by the toxicity of the compound to rats. Diesel fuel is relatively non-toxic to rats based on an acute oral LD<sub>50</sub> (lethal dose for 50 percent of the organisms) of 7,380 mg/kg (Beck et al. 1982) (dose used to calculate TRV).

### **3.3.2 Benzene**

Benzene was identified as a COC in soil/sediments at the Cape Lisburne sites. Benzene is a VOC that is typically a component of fuel oils. Information concerning the toxicity of benzene to the groups of representative species follows.

**3.3.2.1 Plants.** No studies of benzene toxicity to plants were available. Benzene is evaluated as a VOC in Section 3.4.1 (Risk Characterization).

**3.3.2.2 Aquatic Organisms.** Benzene was not identified as a COC in surface water. As a result, no toxicity information for aquatic organisms is presented.

**3.3.2.3 Birds.** No avian toxicity studies were available for benzene. See Section 3.4.3 (Risk Characterization) for a discussion of benzene toxicity to avian species.

**3.3.2.4 Mammals.** Based on oral dose studies of laboratory mice, Nawrot and Staples (1979 in Opresko et al. 1994) report a benzene NOAEL of 26.36 mg/kg. The study evaluated a reproduction endpoint, and this dose is used as a chronic NOAEL to establish the benzene TRV.

### **3.3.3 Toluene**

Toluene was identified as a COC in soil/sediment at the Cape Lisburne sites. It is a VOC and is classed as an alkylbenzene, which is often a component of fuel oils. The following sections present toxicity information for toluene.

**3.3.3.1 Plants.** No studies of toluene toxicity to plants were available. Toluene is evaluated as a VOC in Section 3.4.1 (Risk Characterization).

**3.3.3.2 Aquatic Organisms.** Toluene was not identified as a COC in surface water. As a result, no toxicity information for aquatic organisms is presented.

**3.3.3.3 Birds.** No avian toxicity studies were available for toluene. See Section 3.4.3 (Risk Characterization) for a discussion of toluene toxicity to avian species.

**3.3.3.4 Mammals.** Based on oral dose studies of laboratory mice, Nawrot and Staples (1979 in Opresko et al. 1994) report a toluene NOAEL of 25.98 mg/kg. The study evaluated a reproduction endpoint, and this dose is used as a chronic NOAEL to establish the toluene TRV.

### **3.3.4 Xylene**

Xylene is a COC in soil/sediment at the Cape Lisburne sites. It is a VOC, and most toxicity information in the literature relates to the inhalation of xylene. A summary of the relevant information is presented below.

**3.3.4.1 Plants.** In a study of the green algae, *Selenastrum capricornutum*, xylene decreased growth at concentrations of 72,000 µg/L (Gaur 1988 in AQUIRE 1990).

**3.3.4.2 Aquatic Organisms.** Xylene is not a COC in surface water, so the toxicity to aquatic organisms is not presented.

**3.3.4.3 Birds.** When mallard eggs were immersed in xylene (1 and 10 percent) for 30 seconds, there was no significant effect at concentrations of 10 percent on embryonic weight and length when compared to controls [Hoffman and Eastin 1981 in Hazardous Substance Data Bank (HSDB) 1994]. Japanese quail (*Coturnix japonica*) fed xylene demonstrated no sign of toxicity up to 5,000 ppm (USFWS 1986). The  $LC_{50}$  was >20,000 ppm (USFWS 1986). Hill and Camardese (1986) report a maximum dietary exposure level for Japanese quail of 625 mg/kg total xylenes (dose used to calculate TRV).

**3.3.4.4 Mammals.** Ingestion of xylene in mammals may cause prenatal mortality, growth inhibition, and malformations, primarily cleft palate. The  $LD_{50}$  for ingestion of xylene (rat) was reported as 4,300 mg/kg (Clayton and Clayton 1981) (dose used to calculate TRV).

### **3.3.5 1,4-Dichlorobenzene**

1,4-Dichlorobenzene was identified as a COC in soil/sediment. It readily volatilizes when exposed to air, does not dissolve easily, and may cling to particles of soil and sediment (ATSDR 1991a). This COC was detected only once (sample 2S15, at the Spill/Leak #3 site). The following sections present toxicity information for 1,4-dichlorobenzene.

**3.3.5.1 Plants.** No studies of 1,4-dichlorobenzene toxicity to plants were available. See Section 3.4.1 (Risk Characterization) for a discussion of 1,4-dichlorobenzene toxicity to plants.

**3.3.5.2 Aquatic Organisms.** 1,4-Dichlorobenzene was not found to be a COC in water, so aquatic organisms are not evaluated for exposure to 1,4-dichlorobenzene.

**3.3.5.3 Birds.** No avian toxicity studies were available for 1,4-dichlorobenzene. See Section 3.4.3 (Risk Characterization) for a discussion of 1,4-dichlorobenzene toxicity to avian species.

**3.3.5.4 Mammals.** Hollingsworth et al. (1956 in ATSDR 1991a) report a NOAEL for laboratory rats of 18.8 mg/kg (dose used to calculate the TRV). The endpoints in their study were hepatic and renal effects based on oral doses over a six-month period.

### **3.3.6 4-Methylphenol**

4-Methylphenol was identified as a COC in soil/sediment. 4-Methylphenol is also known as p-cresol. Natural cresols are constantly released to soils via excrement/exocellular secretions and decay of organisms (ATSDR 1991b). Cresols break down rapidly when released to the environment. Under aerobic conditions in soil the half life is expected to be on the order of weeks. This process is likely to be similar in sediments (ATSDR 1991b). The following sections present toxicity information for 4-methylphenol.

**3.3.6.1 Plants.** There is no information available regarding the toxicity of 4-methylphenol to plants. See Section 3.4.1 (Risk Characterization) for a discussion of 4-methylphenol toxicity to plants.

**3.3.6.2 Aquatic Organisms.** 4-Methylphenol was not found to be a COC in water, so it is not quantitatively evaluated in terms of exposure of aquatic organisms.

**3.3.6.3 Birds.** There is no information available concerning the toxicity of 4-methylphenol to birds. See Section 3.4.3 (Risk Characterization) for a discussion of 4-methylphenol toxicity to avian species.

**3.3.6.4 Mammals.** In acute studies, researchers derived an LD<sub>50</sub> of 2,020 mg/kg/day for laboratory rats (ATSDR 1991b). Other studies determined NOAELs for respiratory and hepatic effects of 175 and 450 mg/kg/day, respectively. The TRV used in this risk assessment is based on an LD<sub>50</sub> for rat (oral exposure) of 207 mg/kg/day (Sax and Lewis 1989).

### **3.3.7 Polychlorinated Biphenyls**

PCBs were determined to be a COC in soil/sediment at the Cape Lisburne sites. PCBs are organic compounds commercially produced through the chlorination of a biphenyl molecule (Eisler 1986). Depending upon the chlorination, ten possible congener groups exist. Within the congener groups a number of different isomers exist, each with varying toxicity. The PCBs detected at the Cape Lisburne sites were Aroclor 1254 and Aroclor 1260. Aroclors are groups of PCBs identified by the degree of chlorination (i.e., Aroclor 1260 is predominantly made up of PCBs with more chlorination than Aroclor 1254). PCBs are very stable in the environment, slow to degrade, and bioaccumulative. The following sections summarize the toxicity of PCBs.

**3.3.7.1 Plants.** Very little toxicity information for plants is available in the literature. However, PCBs may inhibit photosynthesis and cell motility in phytoplankton (Eisler 1986). In addition, aquatic plants may provide a route of exposure into the aquatic food chain. An increase (five times) in somatic mutations was noted in terrestrial plants growing on sediments containing mean PCB residues of 26 mg/kg (predominantly Aroclor 1254) (Eisler 1986).

**3.3.7.2 Aquatic Organisms.** PCBs were not found to be a COC in water, so they are not quantitatively evaluated in terms of exposure of aquatic organisms.

**3.3.7.3 Birds.** PCBs are expected to disrupt patterns of growth, reproduction, metabolism, and behavior in sensitive avian species (Eisler 1986). Mourning doves (*Zenaidura macroura carolinensis*) that were fed Aroclor 1254 for 6 weeks exhibited abnormal courtship behavior and reproductive effort. The tested concentrations were 10 or 40 ppm. The researchers suggested that the disrupted reproductive behavior was due to reduced hormone levels (Tori and Peterle 1983 in Eisler 1986). No reproductive effects were observed in mallards (*Anas platyrhynchos*) exposed to 150 ppm (mg/kg feed) Aroclor 1252 in the diet for 12 weeks during egg laying (Haseltine and Prouty 1980), or in mallards exposed to 25 ppm Aroclor 1254 in the diet for at least a month before egg laying (Custer and Heinz 1980). A chronic reproductive LOAEL for ring-necked pheasants of 1.8 mg/kg was reported by Heath et al. (1972). This is the dose used to calculate the TRV.

**3.3.7.4 Mammals.** Reproductive toxicity following chronic or subchronic exposures appears to be the most sensitive toxic endpoint of PCB exposure in mammals. Mink (*Mustela vison*) are particularly susceptible to reproductive effects of PCBs. Mink fed contaminated beef (Aroclor 1254) developed reproductive complications at dietary residue levels as low as 0.64 ppm (Platanow and Karstad 1973). A dietary level LC<sub>50</sub> of 6.7 ppm was reported for an exposure period of 9 months for Aroclor 1254 (Ringer 1983). Other species may be less sensitive to the toxic effects of PCBs. Avlerich and Ringer (1977 in Opresko et al. 1994) report a NOAEL dose of Aroclor 1254 for mink of 0.137 mg/kg. This is used to calculate the carnivore (arctic fox) TRV. White-footed mice (*Peromyscus leucopus*) exposed to PCBs at a concentration of 10 ppm (1.35 mg/kg Aroclor 1254 is the LOAEL dose used to calculate the herbivore TRV) in the diet through the second generation exhibited poor reproductive success, growth and development of organs, but no increased mortality (Linzey 1988 in Opresko et al. 1994).

### **3.3.8 Iron**

Iron is an essential trace element required by both plants and animals. It is a COC in surface water at the Cape Lisburne installation. The following sections summarize the toxicity of iron to the groups of representative species.

**3.3.8.1 Plants.** In a study conducted by Foy et al. (1978 in EPA 1985), concentrations of 100 to 500 ppm soluble iron in soil were toxic to rice.

**3.3.8.2 Aquatic Organisms.** Iron may be a threat in aquatic environments in the form of precipitates that can destroy habitats, coat gills, and inhibit oxygen uptake. The EPA uses 1,000 µg/L as the chronic AWQC protective of aquatic life (dose used to calculate TRV) (EPA 1986c).

**3.3.8.3 Birds.** There are few studies available that address the toxicity of iron to species of wild birds. There was no adverse effect produced in turkeys at concentrations of 440 ppm (Woerpel and Balloun 1964 in NAS 1980). NAS (1980) recommends that the maximum tolerable

level of dietary iron of 1,000 ppm be used for poultry. The 1,000 ppm dose converts to 70.0 mg/kg for a maximum tolerable dietary level for a chicken (dose used to calculate TRV).

**3.3.8.4 Mammals.** At high concentrations, iron is toxic to livestock and interferes with phosphorus metabolism (NAS 1974 in EPA 1976). Cattle fed 477  $\mu\text{g/g}$  iron demonstrated a slight decrease in weight gain; concentrations of 1,677  $\mu\text{g/g}$  of iron produced a significant decline in growth rate (EPA 1985). Shanas and Boyd (1969 in NAS 1980) report an acute  $\text{LD}_{50}$  dose of iron for the rat to be 1,000 mg/kg (dose used to calculate TRV for brown lemming and arctic fox). The maximum tolerable dietary level of 500 ppm of iron for sheep is used to calculate the TRV for caribou (NAS 1980).

### 3.3.9 Manganese

Manganese was identified as a COC in surface water. Manganese is considered to be an essential nutrient for animals (ATSDR 1990), and it is important for growth and reproduction. The toxicity of manganese can be affected by pH and water hardness, and these factors are not known for the surface water at the Cape Lisburne installation.

**3.3.9.1 Plants.** In a four-day study conducted using duckweed (*Lemna minor*), an  $\text{EC}_{50}$  (reduction in growth) was reported of 31,000  $\mu\text{g/L}$  (Wang 1986 in AQUIRE 1990). Lewis et al. (1979) studied the species composition of freshwater phytoplankton populations when exposed to manganese. Population composition was altered at 0.1 mg/L manganese. Soil concentrations of 1,500 to 3,000 mg/kg were reported as phytotoxic to all plant species (Kabata-Pendias and Pendias 1984).

**3.3.9.2 Aquatic Organisms.** In a study conducted by Doudoroff and Katz (1953), brook trout were killed within 24 hours when exposed to concentrations of manganese of 6,250  $\mu\text{g/L}$ . *Daphnia* spp. have a reported 16 percent reproductive impairment in water with concentrations of 4,100  $\mu\text{g/L}$  (Biesinger and Christensen 1972 in Lewis et al. 1979) (dose used to calculate TRV for *Daphnia* spp).

**3.3.9.3 Birds.** Vohra and Kratzer (1968 in NAS 1980) exposed young turkeys to dietary manganese for 21 days. A no observed effect level (NOEL) of 4,080 ppm was derived. The maximum tolerable level of manganese that is recommended by the NAS is 2,000 ppm (250 mg/kg body weight) for poultry. This is the value used to calculate the avian TRV.

**3.3.9.4 Mammals.** When fed 9,000 ppm manganese, sheep demonstrated reduced feed intake (Puls 1988). NAS recommends maximum tolerable levels of 1,000 ppm for cattle (15 mg/kg body weight) and sheep (40 mg/kg body weight). The value for sheep is used to calculate the TRV for caribou. A NOAEL of 930 mg/kg-bw/day is reported for rats in ATSDR (1990). The TRVs for brown lemming and arctic fox are based on the 930 mg/kg dose.

### 3.3.10 Characterization of Effects

In this section, toxicity information is presented for the representative ecological receptors evaluated in the risk characterization of this report (Section 3.4). Potential impacts to aquatic

receptors are evaluated by comparing exposure concentrations to TRVs. Potential impacts to terrestrial wildlife are evaluated for the representative species based on comparisons of estimated exposures to TRVs. TRVs for the representative aquatic species (*Daphnia* spp. at the Cape Lisburne installation) are presented in Table 3-16. Exposure to COCs for the terrestrial representative species is primarily through diet, which may include plants, fish, aquatic invertebrates, soils, and surface water. TRVs are derived for COCs in surface water and soil/sediment. TRVs for the representative bird species are presented in Tables 3-17 and 3-18, and for the representative mammal species in Table 3-19.

**3.3.10.1 Toxicity Reference Values.** TRVs are derived by selecting toxicity values from the literature and extrapolating to the species of concern. Uncertainty factors (UFs) and body scaling factors (SIFs) are used in the extrapolation process as described below.

- (1) The first step is to select an appropriate toxicity value from the scientific literature for each combination of chemical and representative or protected species. Test species most similar to the species of concern are preferred. A secondary emphasis is given to tests conducted over a significant portion of the animal's natural lifespan (e.g., chronic tests) when available.
- (2) The second step is to modify the toxicity value, if necessary, through application of UFs associated with the quality of toxicity data to derive a NOAEL. The highest concentration of a material in a toxicity test that has no statistically significant adverse effect on the exposed population of test organisms as compared with the next highest dose tested. If a chronic NOAEL or NOEL is available, then the toxicity value is used with a UF of one (i.e., no adjustment) because these values have the lowest uncertainty. If chronic data are unavailable, acute or subchronic toxicity data are modified by UFs to extrapolate to chronic effects. Based on Harding Lawson Associates (1992), the following strategy was derived for Ufs for extrapolating study results to chronic NOAELs: 10 for chronic lowest observed effect level (LOEL) values, 10 for subchronic NOEL values, and 20 for subchronic LOEL values.  $LC_{50}$  and  $LD_{50}$  values are extrapolated to chronic NOAELs by a factor of 20.
- (3) The third step is applicable only to terrestrial receptors. This step extrapolates the estimated NOAEL from the test species to a NOAEL for the species of concern using a body SIF. Klaassen et al. (1986) have indicated that dose expressed on a per unit surface area basis may be more appropriate than dose per unit body weight. The underlying assumption is that a toxicant acts on a physiologic surface and that the toxic effect increases as the ratio of chemical to surface area increases. The SIF accounts for differences in the mass to surface area ratios between species. In this assessment the SIF is calculated using the

**TABLE 3-16. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE CAPE LISBURNE INSTALLATION**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	CONCENTRATION (µg/L)	TEST SPECIES	NOAEL UF	INTERSPECIES UF	PROTECTED SPECIES UF	TRV (µg/L)	REFERENCE
Iron	<i>Daphnia</i> spp.	EPA chronic water quality criteria	1,000	all aquatic life	1	1	1	1,000	EPA 1986c
Manganese	<i>Daphnia</i> spp.	reproductive impairment LOAEL	4,100	<i>Daphnia</i> spp.	10	1	1	410	USACOE 1991



TABLE 3-17. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE CAPE LISBURNE INSTALLATION

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Iron	Lapland longspur	NOAEL; 28 day growth study	70	chicken	1	0.32	2	2	54.7	McGhee et al. 1985 in NAS 1980
Iron	brant	NOAEL; 28 day growth study	70	chicken	1	1.18	2	2	14.8	McGhee et al. 1965 in NAS 1980
Iron	glaucous gull	NOAEL; 28 day growth study	70	chicken	1	1.22	2	2	14.3	McGhee et al. 1965 in NAS 1980
Iron	pectoral sandpiper	NOAEL; 28 day growth study	70	chicken	1	0.46	2	2	38.0	McGhee et al. 1965 in NAS 1980
Manganese	Lapland longspur	systemic LOAEL	250	chicken	10	0.32	2	2	19.5	NAS 1980
Manganese	brant	systemic LOAEL	250	chicken	10	1.18	2	2	5.3	NAS 1980
Manganese	glaucous gull	systemic LOAEL	250	chicken	10	1.22	2	2	5.1	NAS 1980
Manganese	pectoral sandpiper	systemic LOAEL	250	chicken	10	0.46	2	2	13.6	NAS 1980

**TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE CAPE LISBURNE INSTALLATION**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
DRPH	Lapland longspur	decreased weight gain LOAEL	6,000	mallard	10	0.29	2	2	517	Hartung 1964
DRPH	brant	decreased weight gain LOAEL	6,000	mallard	10	1.07	2	2	140	Hartung 1964
DRPH	glaucous gull	decreased weight gain LOAEL	6,000	mallard	10	1.10	2	2	136	Hartung 1964
DRPH	pectoral sandpiper	decreased weight gain LOAEL	6,000	mallard	10	0.42	2	2	357	Hartung 1964
Benzene	No avian toxicity data available for benzene; see discussion in Section 3.4.3, Potential Risks to Representative Species of Birds									
Toluene	No avian toxicity data available for toluene; see discussion in Section 3.4.3, Potential Risks to Representative Species of Birds									
Xylenes (total)	Lapland longspur	maximum tolerable dietary level	608	Japanese quail	10	0.60	2	2	25	Hill and Camardese 1986
Xylenes (total)	brant	maximum tolerable dietary level	608	Japanese quail	10	2.16	2	2	7	Hill and Camardese 1986
Xylenes (total)	glaucous gull	maximum tolerable dietary level	608	Japanese quail	10	2.23	2	2	7	Hill and Camardese 1986
Xylenes (total)	pectoral sandpiper	maximum tolerable dietary level	608	Japanese quail	10	0.85	2	2	18	Hill and Camardese 1986
1,4-Dichlorobenzene	No avian toxicity data available for 1,4-Dichlorobenzene; see discussion in Section 3.4.3, Potential Risk to Representative Species of Birds									
4-Methylphenol	No avian toxicity data available for 4-Methylphenol; see discussion in Section 3.4.3, Potential Risks to Representative Species of Birds									

**TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE CAPE LISBURNE INSTALLATION  
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
PCBs (Aroclor 1254)	Lapland longspur	chronic reproductive LOAEL	1.8	ring-necked pheasant	10	0.29	2	2	0.2	Heath et al. 1972
PCBs (Aroclor 1254)	brant	chronic reproductive LOAEL	1.8	ring-necked pheasant	10	1.05	2	2	0.04	Heath et al. 1972
PCBs (Aroclor 1254)	glaucous gull	chronic reproductive LOAEL	1.8	ring-necked pheasant	10	1.08	2	2	0.04	Heath et al. 1972
PCBs (Aroclor 1254)	pectoral sandpiper	chronic reproductive LOAEL	1.8	ring-necked pheasant	10	0.41	2	2	0.1	Heath et al. 1972

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE CAPE LISBURNE INSTALLATION**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
<b>ORGANICS</b>										
DRPH	brown lemming	LD <sub>50</sub>	7,380	rat	20	0.60	2	1	308	Beck et al. 1982
DRPH	arctic fox	LD <sub>50</sub>	7,380	rat	20	2.70	2	1	68	Beck et al. 1982
DRPH	caribou	LD <sub>50</sub>	7,380	rat	20	7.24	2	1	25	Beck et al. 1982
Benzene	brown lemming	reproductive NOAEL	26.36	mouse	1	1.30	2	1	10.1	Opresko et al. 1994
Benzene	arctic fox	reproductive NOAEL	26.36	mouse	1	5.82	2	1	2.3	Opresko et al. 1994
Benzene	caribou	reproductive NOAEL	26.36	mouse	1	15.59	2	1	0.9	Opresko et al. 1994
Toluene	brown lemming	reproductive NOAEL	25.98	mouse	1	1.30	2	1	10.0	Opresko et al. 1994
Toluene	arctic fox	reproductive NOAEL	25.98	mouse	1	5.82	2	1	2.2	Opresko et al. 1994
Toluene	caribou	reproductive NOAEL	25.98	mouse	1	15.59	2	1	0.8	Opresko et al. 1994
Xylenes (total)	brown lemming	LD <sub>50</sub>	4,300	rat	20	0.60	2	1	179	Clayton and Clayton 1981
Xylenes (total)	arctic fox	LD <sub>50</sub>	4,300	rat	20	2.70	2	1	40	Clayton and Clayton 1981
Xylenes (total)	caribou	LD <sub>50</sub>	4,300	rat	20	7.24	2	1	15	Clayton and Clayton 1981
1,4-Dichlorobenzene	brown lemming	hepatic and renal effects NOAEL	18.8	rat	1	0.60	2	1	15.7	Hollingsworth et al. 1956 in ATSDR 1991a
1,4-Dichlorobenzene	arctic fox	hepatic and renal effects NOAEL	18.8	rat	1	2.70	2	1	3.5	Hollingsworth et al. 1956 in ATSDR 1991a

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE CAPE LISBURNE INSTALLATION  
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
1,4-Dichlorobenzene	caribou	hepatic and renal effects NOAEL	18.8	rat	1	7.24	2	1	1.3	Hollingsworth et al. 1956 in ATSDR 1991a
4-Methylphenol	brown lemming	LD <sub>50</sub>	207	rat	20	0.60	2	1	8.6	Sax and Lewis 1989
4-Methylphenol	arctic fox	LD <sub>50</sub>	207	rat	20	2.70	2	1	1.9	Sax and Lewis 1989
4-Methylphenol	caribou	LD <sub>50</sub>	207	rat	20	7.24	2	1	0.7	Sax and Lewis 1989
1,2,4-Trimethylbenzene	brown lemming	LD <sub>50</sub>	8,970	rat	20	0.60	2	1	374	Sax and Lewis 1989
1,2,4-Trimethylbenzene	arctic fox	LD <sub>50</sub>	8,970	rat	20	2.70	2	1	83	Sax and Lewis 1989
1,2,4-Trimethylbenzene	caribou	LD <sub>50</sub>	8,970	rat	20	7.24	2	1	31	Sax and Lewis 1989
PCBs (Aroclor 1254)	brown lemming	reproductive NOAEL	0.135	mouse	1	1.30	2	1	0.05	Opresko et al. 1994
PCBs (Aroclor 1254)	arctic fox	reproductive NOAEL	0.137	mink	1	1.70	2	1	0.04	Opresko et al. 1994
PCBs (Aroclor 1254)	caribou	reproductive NOAEL	0.135	mouse	1	15.59	2	1	0.004	Opresko et al. 1994

**TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE CAPE LISBURNE INSTALLATION  
(CONTINUED)**

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
<b>INORGANICS</b>										
Iron	brown lemming	Acute LD <sub>50</sub>	1,000	rat	20	0.60	2	1	42	Shanas and Boyd 1969 in NAS 1980
Iron	arctic fox	Acute LD <sub>50</sub>	1,000	rat	20	2.70	2	1	9.3	Shanas and Boyd 1969 in NAS 1980
Iron	caribou	NOAEL; maximum tolerable dietary level	20	sheep	1	1.17	2	1	8.5	NAS 1980
Manganese	brown lemming	chronic systemic NOAEL	930	rat	1	0.60	2	1	780	Hejtmanick et al. 1987 in ATSDR 1990
Manganese	arctic fox	chronic systemic NOAEL	930	rat	1	2.7	2	1	170	Hejtmanick et al. 1987 in ATSDR 1990
Manganese	caribou	NOAEL; maximum tolerable dietary level	40	sheep	1	1.17	2	1	17	NAS 1980

following equation (Mantel and Schneiderman 1975) (SIFs are presented in Appendix F):

$$\text{SIF} = (\text{weight of representative species/weight of test species})^{1/3}.$$

- (4) A UF of 2 is used to account for interspecies variation in sensitivity. This value is based on the methodology used by Harding Lawson Associates (1992).
- (5) A UF of 2 was used to account for additional sensitivity of state and/or federally protected species. This value is based on Harding Lawson Associates (1992). Migratory birds are federally protected and include all the representative avian and protected species selected for this assessment.

The methods of calculating the TRV for the terrestrial and aquatic receptors are as follows:

#### **TERRESTRIAL:**

- a) Convert test dose to a NOAEL:

$$\text{DOSE} \div \text{NOAEL UF} = \text{Estimated NOAEL}$$

- b) Adjust for body size difference between test species and ROC:

$$\text{Estimated NOAEL} \div \text{SCALING FACTOR} = \text{Scaled, estimated NOAEL}$$

- c) Adjust for interspecific differences:

$$\text{Scaled, estimated NOAEL} \div \text{INTERSPECIES UF} = \text{Species-specific, scaled, estimated NOAEL}$$

- D) Account for protected species status:

$$\text{Species-specific, scaled, estimated NOAEL} \div \text{PROTECTED SPECIES UF} = \text{TRV}$$

#### **AQUATIC:**

$$\text{EFFECTIVE CONCENTRATION} \div \text{NOAEL UF} \div \text{INTERSPECIES UF} = \text{TRV}$$

### **3.4 RISK CHARACTERIZATION FOR ECOLOGICAL RECEPTORS**

In this section, potential risks to ecological receptors (representative species) are documented. Potential risks to plants were evaluated based on the contaminant concentrations in the soil/sediment and information from the literature. Potential risks to aquatic organisms, birds, and mammals were estimated by comparing estimated exposures to TRVs (i.e., quotient method). The quotient method divides the estimated exposure concentration by the associated TRV to derive the HQ. If the HQ is less than 1.0, then adverse effects are not expected. Conversely, if the HQ is equal to or greater than 1.0, a potential for adverse effects exists. The confidence level of the risk estimate is increased as the magnitude of the HQ departs from 1.0. For example, there is greater confidence in a risk estimate where the HQ is 0.1 or 10, than in a HQ such as

0.9 or 1.1. The confidence level is also dependent on the uncertainty associated with the estimated exposure and the TRV for a given chemical-receptor combination.

The characterization of risk focuses on the assessment endpoints. These endpoints were selected and discussed in keeping with the Framework for Ecological Risk Assessment guidance (EPA 1992a). The assessment endpoints for the Cape Lisburne ERA are changes in:

- the populations of the plant representative species (*Carex* spp., *Salix* spp., *Eriophorum* spp., and *Vaccinium* spp.);
- the populations of the aquatic representative species (*Daphnia* spp.);
- the populations of avian representative species (Lapland longspur, brant, glaucous gull, and pectoral sandpiper); and
- the populations of mammalian representative species (brown lemming, arctic fox, and barren-ground caribou).

The measurement endpoints used to evaluate potential changes in populations of the representative species were based on the endpoints used to derive the TRVs. These endpoints included physiological effects, growth, reproduction, and mortality.

Potential ecological risks are presented in the following sections: Section 3.4.1 addresses representative species of plants; Section 3.4.2 considers aquatic organisms; Section 3.4.3 addresses representative species of birds; and Section 3.4.4 discusses representative species of mammals. A discussion of potential future risks to ecological receptors is presented in Section 3.4.5. The HQs that represent potential risk estimates are summarized in Tables 3-21 and 3-23.

#### **3.4.1 Potential Risks to Representative Species of Plants**

In determining the risks to plants at the Cape Lisburne sites, a qualitative comparison was made of soil and surface water contaminant concentrations and plant toxicity information in the literature. Table 3-20 summarizes these comparisons. Little information is available regarding the toxicity of the Cape Lisburne COCs in soil/sediment (only PCB toxicity data are shown in Table 3-20), although the organic COCs are less bioavailable in soil/sediment than COCs in surface waters. There is a great deal of uncertainty in this phase of the assessment because of the differences in degree of uptake between plant species (Walker et al. 1978). However, the concentrations of contaminants onsite can be compared on the level of orders of magnitude.

This comparison allows broad trends to be observed in order to determine whether a potential risk may exist.

Information is generally lacking concerning the toxicity of the COCs at Cape Lisburne and how they relate to the representative species of plants. As a result, when comparisons of TRVs for site-specific species and chemicals are not possible, comparisons of related chemicals with other



**TABLE 3-20. COMPARISON OF COC CONCENTRATIONS TO TOXICITY INFORMATION FOR PLANTS**

CHEMICAL (COC media)	PLANT	EXPOSURE LEVEL	EFFECT ON PLANT	CAPE LISBURNE EXPOSURE	REFERENCE
IRON (COC in water)	rice	100,000 - 500,000 µg/L >500,000 µg/L	toxic; highly toxic	2,405 µg/L	Foy et al. in EPA 1985
MANGANESE (COC in water)	duckweed	31,000 µg/L in water	EC <sub>50</sub>	670 µg/L	USACOE 1991
VOCs (COCs in soil/sediment)	green algae	4,600 µg/L for ethylbenzene 2,290 µg/L for methylene chloride, in water	EC <sub>50</sub>	none in water, 0.08 mg/kg as benzene 0.43 mg/kg as toluene in soil/sediments	USACOE 1991
PCBs (Aroclor 1254) (COC in soil/sediment)	pigweed	40 mg/kg 20 mg/kg	22% weight reduction NOAEL	28 mg/kg as 1254 274 mg/kg as 1260	Strek and Weber 1982 in Will and Suter 1994

**TABLE 3-21. RISK CHARACTERIZATION OF REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE CAPE LISBURNE INSTALLATION**

SPECIES	ESTIMATED EXPOSURE CONCENTRATION (µg/L)	TRV (µg/L)	HAZARD QUOTIENT
<b>IRON</b>			
<i>Daphnia</i> spp.	2,405	1,000	2.4
<b>MANGANESE</b>			
<i>Daphnia</i> spp.	670	410	1.6
<b>1,2,4-TRIMETHYLBENZENE</b>			
<i>Daphnia</i> spp.	0.6	NA	NC

NA = toxicity information not available.  
NC = HQ not calculated because no TRV available.

**TABLE 3-22. RELATIVE TOXICITY RANKINGS FOR COMPARISON OF MAMMALIAN AND AVIAN TOXICITY**

COC	STUDY TYPE*	DOSE* (mg/kg)	RELATIVE TOXICITY
benzene	oral rat LD <sub>50</sub>	3,400	slightly toxic
toluene	oral rat LD <sub>50</sub>	5,000	slightly toxic
1,4-dichlorobenzene	oral rat LD <sub>50</sub>	500	moderately toxic
4-methylphenol	oral rat LD <sub>50</sub>	207	moderately toxic
1,2,4-trimethylbenzene	oral rat LD <sub>50</sub>	8,970	relatively nontoxic

\* Sax and Lewis 1989.

**TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE CAPE LISBURNE INSTALLATION**

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
<b>IRON</b>			
Lapland longspur	$5 \times 10^{-1}$	55	$8 \times 10^{-3}$
brant	$7 \times 10^{-2}$	15	$5 \times 10^{-3}$
glaucous gull	$1 \times 10^{-2}$	14	$9 \times 10^{-4}$
pectoral sandpiper	$3 \times 10^{-1}$	38	$8 \times 10^{-3}$
brown lemming	$2 \times 10^{-1}$	42	$4 \times 10^{-3}$
arctic fox	$6 \times 10^{-3}$	9.3	$7 \times 10^{-4}$
caribou	$2 \times 10^{-3}$	8.5	$2 \times 10^{-4}$
<b>MANGANESE</b>			
Lapland longspur	$1 \times 10^{-1}$	20	$6 \times 10^{-3}$
brant	$2 \times 10^{-2}$	5	$4 \times 10^{-3}$
glaucous gull	$3 \times 10^{-3}$	5	$7 \times 10^{-4}$
pectoral sandpiper	$8 \times 10^{-2}$	14	$6 \times 10^{-3}$
brown lemming	$4 \times 10^{-2}$	780	$5 \times 10^{-5}$
arctic fox	$2 \times 10^{-3}$	170	$1 \times 10^{-5}$
caribou	$4 \times 10^{-4}$	17	$3 \times 10^{-5}$
<b>DRPH</b>			
Lapland longspur	$3 \times 10^1$	520	$6 \times 10^{-2}$
brant	$2 \times 10^1$	140	$1 \times 10^{-1}$
glaucous gull	$2 \times 10^0$	140	$1 \times 10^{-2}$
pectoral sandpiper	$1 \times 10^2$	360	$3 \times 10^{-1}$
brown lemming	$1 \times 10^2$	310	$4 \times 10^{-1}$
arctic fox	$2 \times 10^{-1}$	69	$3 \times 10^{-3}$
caribou	$6 \times 10^{-2}$	26	$2 \times 10^{-3}$
<b>BENZENE</b>			
Lapland longspur	$1 \times 10^{-2}$	NA	NC
brant	$5 \times 10^{-3}$	NA	NC

NA = Toxicity information not available. See text for qualitative discussion.

NC = Not able to calculate because no TRV available. See text for qualitative discussion.

**TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE CAPE LISBURNE INSTALLATION (CONTINUED)**

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
glaucous gull	$1 \times 10^{-4}$	NA	NC
pectoral sandpiper	$5 \times 10^{-3}$	NA	NC
brown lemming	$8 \times 10^{-2}$	10	$8 \times 10^{-3}$
arctic fox	$4 \times 10^{-6}$	2.3	$2 \times 10^{-6}$
caribou	$5 \times 10^{-5}$	0.9	$5 \times 10^{-5}$
<b>TOLUENE</b>			
Lapland longspur	$3 \times 10^{-2}$	NA	NC
brant	$1 \times 10^{-2}$	NA	NC
glaucous gull	$4 \times 10^{-4}$	NA	NC
pectoral sandpiper	$2 \times 10^{-2}$	NA	NC
brown lemming	$2 \times 10^{-1}$	10	$2 \times 10^{-2}$
arctic fox	$2 \times 10^{-5}$	2.2	$9 \times 10^{-6}$
caribou	$1 \times 10^{-4}$	0.8	$2 \times 10^{-4}$
<b>XYLENES (total)</b>			
Lapland longspur	$5 \times 10^{-2}$	26	$2 \times 10^{-3}$
brant	$2 \times 10^{-2}$	7.2	$3 \times 10^{-3}$
glaucous gull	$7 \times 10^{-4}$	6.8	$1 \times 10^{-4}$
pectoral sandpiper	$3 \times 10^{-2}$	18	$2 \times 10^{-3}$
brown lemming	$3 \times 10^{-1}$	180	$2 \times 10^{-3}$
arctic fox	$4 \times 10^{-5}$	40	$9 \times 10^{-7}$
caribou	$2 \times 10^{-4}$	15	$1 \times 10^{-5}$
<b>1,4-DICHLOROBENZENE</b>			
Lapland longspur	$2 \times 10^{-3}$	NA	NC
brant	$8 \times 10^{-4}$	NA	NC
glaucous gull	$3 \times 10^{-5}$	NA	NC
pectoral sandpiper	$2 \times 10^{-3}$	NA	NC
brown lemming	$1 \times 10^{-2}$	16	$7 \times 10^{-4}$

NA = Toxicity information not available. See text for qualitative discussion.

NC = Not able to calculate because no TRV available. See text for qualitative discussion.

**TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE CAPE LISBURNE INSTALLATION (CONTINUED)**

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
arctic fox	$3 \times 10^{-6}$	3.5	$7 \times 10^{-7}$
caribou	$7 \times 10^{-6}$	1.3	$5 \times 10^{-6}$
<b>4-METHYLPHENOL</b>			
Lapland longspur	$7 \times 10^{-1}$	NA	NC
brant	$3 \times 10^{-1}$	NA	NC
glaucous gull	$7 \times 10^{-3}$	NA	NC
pectoral sandpiper	$3 \times 10^{-1}$	NA	NC
brown lemming	$5 \times 10^0$	8.6	$6 \times 10^{-1}$
arctic fox	$2 \times 10^{-4}$	1.9	$9 \times 10^{-5}$
caribou	$3 \times 10^{-3}$	0.7	$4 \times 10^{-3}$
<b>1,2,4-TRIMETHYLBENZENE</b>			
Lapland longspur	$1 \times 10^{-4}$	NA	NC
brant	$2 \times 10^{-5}$	NA	NC
glaucous gull	$3 \times 10^{-6}$	NA	NC
pectoral sandpiper	$8 \times 10^{-5}$	NA	NC
brown lemming	$4 \times 10^{-5}$	374	$1 \times 10^{-7}$
arctic fox	$2 \times 10^{-6}$	83	$2 \times 10^{-8}$
caribou	$4 \times 10^{-7}$	31	$1 \times 10^{-8}$
<b>AROCLOR 1254</b>			
Lapland longspur	$1 \times 10^{-1}$	0.2	$7 \times 10^{-1}$
brant	$7 \times 10^{-2}$	0.04	$2 \times 10^0$
glaucous gull	$1 \times 10^{-2}$	0.04	$2 \times 10^{-1}$
pectoral sandpiper	$7 \times 10^{-1}$	0.1	$7 \times 10^0$
brown lemming	$4 \times 10^{-1}$	0.05	$7 \times 10^0$
arctic fox	$1 \times 10^{-3}$	0.04	$3 \times 10^{-2}$
caribou	$2 \times 10^{-4}$	0.004	$4 \times 10^{-2}$

NA = Toxicity information not available. See text for qualitative discussion.  
 NC = Not able to calculate because no TRV available. See text for qualitative discussion.

**TABLE 3-23. HAZARD QUOTIENTS FOR REPRESENTATIVE BIRDS AND MAMMALS AT THE CAPE LISBURNE INSTALLATION (CONTINUED)**

CHEMICAL OF CONCERN	ESTIMATED EXPOSURE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
<b>AROCLOR 1260</b>			
Lapland longspur	$1 \times 10^0$	0.2	$7 \times 10^0$
brant	$7 \times 10^{-1}$	0.04	$2 \times 10^1$
glaucous gull	$1 \times 10^{-1}$	0.04	$2 \times 10^0$
pectoral sandpiper	$7 \times 10^0$	0.1	$7 \times 10^1$
brown lemming	$4 \times 10^0$	0.05	$7 \times 10^1$
arctic fox	$1 \times 10^{-2}$	0.04	$3 \times 10^{-1}$
caribou	$2 \times 10^{-3}$	0.004	$4 \times 10^{-1}$

NA = Toxicity information not available. See text for qualitative discussion.  
 NC = Not able to calculate because no TRV available. See text for qualitative discussion.

plant species are made. As seen in Table 3-20, the concentrations of iron and manganese found in the surface water at Cape Lisburne are over 40 times lower than reported toxicity values. In addition, the concentrations of VOCs at the site are substantially lower than toxicity values reported by Galassi et al. (1988 in USACOE 1991) and Hutchinson et al. (1980 in USACOE 1991). Note that the toxicity values are given for VOCs in solution, and the VOCs of concern at Cape Lisburne are in soil/sediment. However, comparing the water concentrations ( $\mu\text{g/L}$  or ppb) to the soil/sediment concentrations (mg/kg or ppm) shows that the concentrations at Cape Lisburne are below the toxicity levels. The VOCs are not expected to be present at significant levels in most plants because of the VOCs' volatility, absorption to soil particles, use in the plant's metabolic processes, and degradation rates in soil (Kostecki and Calabrese 1989).

The risk to plants from PCBs is estimated to be moderate at the specific locations that the PCB concentrations were elevated. However, these locations are generally not conducive to plant growth, regardless of COCs that may be present. Several sample locations were within structures, and the vast majority of samples were taken on the gravel pads where the lack of organic matter severely limits plant growth. The moderate risk to plants posed by PCBs should not be overlooked at the specific sample locations, but the overall risk to plants from organic COCs over the entire Cape Lisburne installation is estimated to be low.

The risk to plants from 1,4-dichlorobenzene and 4-methylphenol cannot be quantified because no toxicity information is available. However, each of these COCs was detected only once (1/14 for 1,4-dichlorobenzene and 1/6 for 4-methylphenol) and it is likely that each of these COCs will be partially bound to soil/sediment particles and not be bioavailable to plants (ATSDR 1991a, 1991b). It is not expected that the single locations containing the representative detected COCs pose a risk to plant populations at the Cape Lisburne installation.

The comparisons presented in Table 3-20 are not definitive in judging the toxicity of metals to the specific representative plant species; however, noting the differences between exposure concentrations that pose risks and the concentrations at the installation, risks to *Carex*, *Salix*, *Eriophorum*, and *Vaccinium* spp. are likely to be low.

### 3.4.2 Potential Risks to Representative Species of Aquatic Organisms

Estimates of exposure for aquatic organisms were based on the average concentrations of each COC in surface water samples (Section 3.1). The TRVs for aquatic species are presented in Table 3-16. The HQs are calculated by dividing the estimated exposure concentration by the TRV. Table 3-21 presents the results of the risk characterization for aquatic organisms. The following paragraph summarizes the potential risks to aquatic organisms from iron, manganese and 1,2,4-trimethylbenzene, the only COCs identified in surface water.

The HQs for *Daphnia* spp. for iron and manganese in surface water were 2.4 and 1.6, respectively. These HQs indicate that a potential risk to aquatic organisms could exist from iron and manganese concentrations in surface water. These risks are mitigated, however, by the fact that "total" metal concentrations were used to estimate risk. The HQs are based on the average total concentrations of iron (2,405  $\mu\text{g/L}$ ) and manganese (670  $\mu\text{g/L}$ ); the dissolved concentrations (810  $\mu\text{g/L}$  for iron and 337  $\mu\text{g/L}$  for manganese) are not expected to pose risks to *Daphnia* spp.

No significant risks are expected from 1,2,4-trimethylbenzene because this COC may be ranked as relatively nontoxic (Smith 1987). See the following section for a discussion of the relative ranking of toxicities. Based on the available data and the discussions presented, the risk to aquatic organisms at the five Cape Lisburne sites is not estimated to be significant.

### 3.4.3 Potential Risks to Representative Species of Birds

The HQs for Aroclor 1254 and Aroclor 1260 are greater than 1.0 for several species of birds. The toxicity of Aroclor 1254 is slightly greater than that of Aroclor 1260 (Eisler 1986), but the avian representative species were evaluated using the more conservative Aroclor 1254 TRV. The resulting risk estimates associated with PCBs at the Cape Lisburne sites are estimated to be low to moderate for the glaucous gull and Lapland longspur, and moderate for the brant and pectoral sandpiper. Some important factors should be considered when reviewing these risk estimates. For example, it is important to note that a portion of the birds' diets (i.e., invertebrates) was not quantified in this assessment. This may result in an underestimate of potential exposure to metals, although iron and manganese were the only metals identified as COCs, and they are not expected to bioaccumulate to levels that would pose any risk to birds.

Quantitative evaluation of benzene, toluene, 1,4-dichlorobenzene, 4-methylphenol, and 1,2,4-trimethylbenzene is not possible because avian toxicity studies for these COCs are not available. However, a discussion of the relative toxicity of these chemicals provides a basis for making qualitative statements concerning their toxicity to avian species. Smith (1987) provides the following ranking of relative toxicities based on median lethal doses ( $LD_{50}$ s).

- I. Extremely toxic ( $LD_{50} \leq 40$  mg/kg)
- II. Highly toxic ( $LD_{50}$  41 - 200 mg/kg)
- III. Moderately toxic ( $LD_{50}$  201 - 1,000 mg/kg)
- IV. Slightly toxic ( $LD_{50}$  1,001 - 5,000 mg/kg)
- V. Relatively nontoxic ( $LD_{50} > 5,000$  mg/kg)

Table 3-22 shows the relative toxicity rankings of benzene, toluene, 1,4-dichlorobenzene, 4-methylphenol, and 1,2,4-trimethylbenzene, based on the ranking scheme of Smith (1987).

It is possible to make inferences concerning the potential risk to avian species using the relative toxicities of these COCs, the exposure estimates for avian and mammalian species, and the HQs calculated for mammalian species (see Table 3-23 for exposure estimates and HQs).

There is limited information available on the relative toxicologic sensitivities of birds compared to mammals. Based upon a review of the species and chemicals tested (Smith 1987; Hudson et al. 1979; and Tucker and Leitzke 1979), it appears in general that avian and mammalian sensitivities (via oral exposure) fall within the same range, with birds overall being slightly more sensitive than mammals. There are, of course, exceptions to this general observation and, for a number of chemicals, mammals are more sensitive than birds. For those cases where birds are more sensitive, most avian toxicity values fall well within one order of magnitude of the mammalian toxicity values.



Avian toxicity values are not available for some of the COCs in this ERA. The HQs for mammals range from  $1 \times 10^{-8}$  (caribou exposure to 1,2,4-trimethylbenzene) to  $6 \times 10^{-1}$  (lemming exposure to 4-methylphenol). The avian estimated exposures for these COCs are within the range of mammalian estimated exposures (avian range of  $3 \times 10^{-6}$  to  $7 \times 10^{-1}$ , mammalian range of  $2 \times 10^{-6}$  to 5). The greatest estimated avian exposure for 4-methylphenol (ranked moderately toxic) is  $7 \times 10^{-1}$  mg/kg (Lapland longspur), while the brown lemming's estimated exposure to 4-methylphenol is 5 mg/kg. For 1,4-dichlorobenzene, also ranked as moderately toxic, the greatest avian estimated exposure (Lapland longspur) is  $2 \times 10^{-3}$  mg/kg, which is less than the lemming's estimated exposure of  $1 \times 10^{-2}$  mg/kg. This results in qualitative avian risk estimates for the moderately toxic COCs (i.e., 1,4-dichlorobenzene and 4-methylphenol) that are less than the mammalian risk estimates (i.e., HQs less than 1.0). Because the estimated exposures for avian species for respective COCs are less than the mammalian estimated exposures, risk estimates for benzene and toluene, ranked as slightly toxic, and for 1,2,4-trimethylbenzene, ranked as relatively nontoxic, are commensurately less than the mammalian risk estimates, which are all less than 1.0.

Based on the information presented above, these chemicals are not expected to be significantly more toxic to birds than to mammals. Birds are not expected to be at risk given that there are no HQs above one for mammals, and the estimated exposures for birds are sufficiently less than those for mammals to offset the possibility that some birds may be more sensitive than mammals to these selected COCs.

Note that this qualitative discussion and evaluation of potential risk introduces an element of uncertainty to the risk assessment. See Section 3.5, Uncertainty Analysis, for more discussion of this topic.

#### **3.4.4 Potential Risks to Representative Species of Mammals**

HQs for the brown lemming were above 1.0 only for Aroclor 1254 and Aroclor 1260, indicating potential risk from these COCs. The HQs for the arctic fox and the barren-ground caribou were below 1.0 for all COCs. Thus, the risk estimates for the arctic fox and barren-ground caribou are not significant at the Cape Lisburne installation.

The toxicity of Aroclor 1254 is slightly greater than that of Aroclor 1260 (Eisler 1986), but the mammalian representative species were evaluated against the stricter Aroclor 1254 standard. The resulting risk estimates for the brown lemming are moderate for the both Aroclor 1254 and Aroclor 1260.

Refer to Section 3.5, Uncertainty Analysis, for a more detailed discussion of the uncertainties associated with these risk estimates.

#### **3.4.5 Potential Future Risks**

Future risk estimates at the Cape Lisburne installation are based on the assumption that all buildings will be removed from the five sites and that the gravel pads will remain in place. Future risks at the Spill/Leak #3 (ST07) are expected to be as low as or lower than current risks (which

are estimated not to be significant) because the exposure pathways are not likely to change, and the concentrations of COCs at Spill/Leak #3 (ST07) are likely to diminish over time. Because soil/sediment samples indicate the presence of PCBs (Aroclor 1254 and/or Aroclor 1260) at the Landfill and Waste Accumulation Area (LF01), White Alice Site (SS03), Upper Camp Transformer Building (SS08), and Lower Camp Transformer Buildings (SS09), future risk estimates may be greater than the current estimates because of the high potential for PCBs to bioaccumulate.

PCBs have very high chemical, thermal, and biological stability in addition to low vapor pressure (Manahan 1994). Conversion of highly substituted PCBs to molecules with one or two chlorines is done relatively slowly by anaerobic bacteria (Manahan 1994). Therefore, natural biodegradation may not be an effective process for significantly reducing the PCBs to concentrations that are protective of the environment. In addition, PCBs have a high potential for bioaccumulation, which may result in a magnification of PCB concentrations and resultant exposure to PCBs by ecological receptors through the food chain.

Risk estimates related to potential exposure to PCBs under current conditions are low at the Landfill and Waste Accumulation Area (LF01), and low to moderate at the White Alice Site (SS03), Upper Camp Transformer Building (SS08), and the Lower Camp Transformer Buildings (SS09). There is potential for all of these estimates to increase over time under the assumption that the buildings will be removed and no remediation is conducted at the site. This scenario would allow potential pathways for exposure to PCBs to be complete.

### **3.5 ECOLOGICAL RISK ASSESSMENT UNCERTAINTY ANALYSIS**

As with any risk assessment, there is great uncertainty associated with the estimates of ecological risk for the sites at the Cape Lisburne installation. The risk estimates are based on a number of assumptions regarding exposure and toxicity. In general, the primary sources of uncertainty are the following:

- Environmental sampling and analysis;
- Selection of COCs;
- Selection of representative species;
- Exposure parameter estimation; and
- Toxicological data.

A complete understanding of the uncertainties associated with risk estimates is critical to placing the predicted risks in proper perspective. The most significant sources of uncertainty associated with the estimates of risk for the Cape Lisburne installation sites are summarized in the following sections.

#### **3.5.1 Environmental Sampling and Analysis**

The principal source of uncertainty in the analytical data (for the ERA) stems from the sampling approach and the subsequent calculation of exposure concentrations. Sampling at the Cape Lisburne installation was conducted in a systematic manner, designed to characterize localized

contaminated areas or "hot spots". The site's potential source areas are, therefore, well characterized; however, there are limited data regarding the peripheral areas (areas to which ecological receptors are most likely to be exposed). In order to compensate for this non-random sampling methodology in the calculation of exposure concentrations, the exposure assessment used the average concentration of COCs across the site.

The methods of calculating the average concentrations were the same for organic and inorganic data. In calculating the average concentration of chemicals at the site, non-detected chemicals were entered at one-half of the quantitation limit, as per EPA guidance (EPA 1989). As noted, sampling was designed to characterize "hot spots" at each of the sites. Therefore, the average concentrations of COCs tend to be biased high because sampling was generally concentrated in areas of the site where significant contamination exists or was suspected. The use of total metal concentrations in surface water to estimate risk is a conservative approach because dissolved metal concentrations are generally significantly less than total metal concentrations. Therefore, the average concentrations of total metals used to estimate exposure in surface water may overestimate potential risk.

The limited analysis of metals is a factor that also contributes to uncertainty. Of the eight surface water samples, only four were analyzed for metals, and metals analysis was performed on only four of the seventy-nine soil/sediment samples used in the ERA. The selection of the inorganic COCs was based on these analyses, and the resultant choices may under- or overestimate the true nature and extent of metals contamination at the Cape Lisburne installation. It is difficult to assess the degree of impact that this uncertainty has on the overall risk estimates, although the available data indicate that metals contamination is not likely to present risks at Cape Lisburne.

An additional factor related to the analysis of surface water samples should be considered. The sample quantitation limits of several metals were higher than the action levels used to screen the chemicals. Therefore, non-detected concentrations of beryllium, cadmium, copper, chromium, lead, selenium, silver, and thallium may be present in quantities sufficient to elicit adverse effects in aquatic organisms. This probably contributes a low level of uncertainty to the overall risk estimate because surface water pathways at the Cape Lisburne installation are not likely to be significant routes of exposure to representative species other than *Daphnia* spp.

Further, there is uncertainty inherent in using measurements of DRPH, GRPH, and RRPH for risk assessments. The analytical techniques are not specific to petroleum (i.e., they detect other organics, including naturally-occurring ones) (Von Burg 1993). Moreover, the toxicity of these groups of petroleum hydrocarbons is determined by the toxicity of their individual constituents. When petroleum compounds are released to the environment, they tend to weather or transform readily. For example, the lighter fractions (such as BTEX) will volatilize to the atmosphere more readily than the heavier fractions (such as decane, pyrene, or benzo(a)pyrene). The lighter fractions are thought to be the more toxic (Wong et al. 1981; O'Brien 1978; Kauss and Hutchinson 1975; and Soto et al. 1975). Therefore, the toxicities of DRPH, GRPH, and RRPH are expected to change over time depending upon the attenuation mechanisms occurring in the environment. As a result, the toxicity of the petroleum hydrocarbons detected at the Cape Lisburne sites is uncertain. Use of toxicity values reported in the literature probably contributes

to an overestimation of the risk because it is likely that the most toxic components of the mixtures detected have volatilized to the atmosphere over time.

### **3.5.2 Selection of Chemicals for Evaluation**

The selection of COCs in the ERA was based upon a comparison to background concentrations and action levels, and an evaluation of the frequency of detection. For certain chemicals, no action levels were available. As a result, action levels for related compounds were used. This introduces some uncertainty into the risk assessment as there is a chance that actual toxicity is different from the toxicity of the surrogate chemical. Overall, however, the process provided a conservative screening of the COCs, and it is unlikely that any chemicals presenting an ecological risk were omitted.

### **3.5.3 Selection of Representative Species**

The selection of representative species in the ERA also introduces some uncertainty into the risk estimates. No site-specific biological surveys were conducted at the Cape Lisburne installation, with the exception of a survey for spectacled and Steller's eiders (Alaska Biological Research 1994). As a result, it is not known whether or how often the representative species are actually found at the site. However, the uncertainty introduced into the risk estimate by this route is low. The purpose of ERAs is not to survey the biota at a site, but to estimate the risks to the species, communities and ecosystems in the area. Surrogate species are, therefore, commonly used. As a result, even if the representative species do not reside specifically at the Cape Lisburne installation, the risk estimates in this report will provide a sound measure of the potential risks to the species that do inhabit the area.

### **3.5.4 Exposure Assessment**

Exposures were estimated from literature-based life history information for the selected representative species. There is moderate uncertainty associated with the exposure information. Food and water ingestion rates were not available for some species and were estimated using regression equations. Incidental ingestion of soils/sediments may occur while animals are foraging in these media, and it is uncertain how much soil/sediment is actually ingested. In addition, there is uncertainty associated with the habitat associated at the site. Samples were collected around buildings and other structures that are likely to provide habitat of limited quality or utility to wild species. As a result, this tends to overestimate exposure. Further, there are significant uncertainties associated with the estimates of how extensively a receptor will use the site, which were based on home range information. As noted in the discussion of Estimation of Percent Ingested Onsite, Section 3.2.7.2, the conversion of population density values as substitutes for home ranges adds uncertainty to the risk assessment. The conversion was necessary because home range data are lacking for some of the representative species.

There is some uncertainty associated with the diet compositions estimated from information obtained from the literature. A good example of this type of uncertainty is the unpredictable fluctuation in the populations of the brown lemmings and their predators (i.e., arctic fox, glaucous gull). As the numbers of prey increase, predator populations may experience numerical and

density increases well beyond the values reported in the literature. When prey populations decrease, predation pressure can shift to diet items that are not considered "normal", and do not represent dietary intakes reported in the literature. Wildlife, and their interactions with the environment, are dynamic. Stochastic events, natural or anthropogenic, may cause behavior and/or habits to differ markedly from the "expected or norm". Deviations from typical behavior cause uncertainty when evaluating wildlife and ecosystems.

There is also uncertainty associated with exposure estimates for plants. Plant uptake of COCs was derived from a regression equation using the  $K_{ow}$  of the COC (Appendix C). This calculation estimates the concentration of chemicals in the vegetative portion of plants. Actual concentrations of the COC in plant tissue will vary depending upon actual chemical uptake, species of plant, and other site-specific factors (such as soil organic carbon). It is important to note that screening level tissue concentrations in plants were not available for comparison with these estimated concentrations. As a result, it is uncertain whether the concentrations are phytotoxic. However, the overall effect of this source of uncertainty in the risk assessment is low, as is the ecological risk to plants.

In addition, the only component in the diet of representative species evaluated quantitatively was the ingestion of plants. Ingestion of animal prey (e.g., the diet of the arctic fox and the insectivorous portion of some avian diets) was not quantified because tissue concentration data were not available. This may slightly underestimate risk for those species that rely on animal items in their diet.

### 3.5.5 Toxicological Data

One of the largest sources of uncertainty in risk assessment is from the toxicological data. Often there are no relevant studies for the specific representative species or endpoints. As a result, extrapolations are made, which introduce uncertainty into the risk estimate. These extrapolations incorporate UFs into the calculation of TRVs. The purpose of the UFs is to incorporate some margin of error into the risk estimate, in order to arrive at a "safe" level of exposure to which estimated exposure concentrations may be compared. These techniques introduce into the risk assessment a tendency to overestimate rather than underestimate the risk, as conservative estimates were made in estimating toxicity values.

For some chemicals, no toxicity information was available (e.g., benzene and toluene for birds). As a result, these compounds were not evaluated quantitatively in the risk assessment, and the risks may be somewhat underestimated. However, based on the low concentrations of these compounds (reported in Section 3.1), the uncertainty associated with this factor is low.

Toxicity values for plants, water, soils, and sediments are based on literature values. Toxicity in soils and sediments is affected by the bioavailability of a given chemical. Toxicity of metals in water is based, in part, upon the speciation of the element. As a result, site-specific bioavailability or toxicity may differ from that in the studies used to estimate potential toxic effects. Therefore actual toxicities of chemicals at the Cape Lisburne sites may be different from the values reported in the literature. In addition, the sensitivity of receptors onsite may be different

from the sensitivity of the species reported in the literature. This contributes to the overall uncertainty of the risk assessment.

There is a great deal of uncertainty in assessing the toxicity of chemical mixtures. In this ERA, the effects of exposure from each contaminant have been considered separately. However, these substances occur together at the site, and organisms may be exposed to mixtures of the chemicals. Prediction of how these mixtures of toxicants will interact must be based on an understanding of the mechanisms of such interactions. The interactions of the individual components of chemical mixtures may occur during absorption, distribution, metabolism, excretion, or activity at the receptor site. Individual compounds may interact chemically, yielding a new toxic component or causing a change in the biological availability of an existing component, or may interact by causing different effects at different receptor sites. Suitable data are not currently available to characterize the effects of chemical mixtures rigorously, so chemicals present at the site were evaluated independently. This approach of assessing risk associated with mixtures of chemicals does not account for any additive, synergistic, or antagonistic interactions among the chemicals considered. However, as discussed in Section 3.6, the risk assessment yielded a low potential for ecological risks (with the exception of risks from PCBs), and it is unlikely that additive effects of chemicals are a concern.

### **3.6 SUMMARY OF ECOLOGICAL RISK**

The potential risks to ecological receptors are summarized in this section based on the information presented in the ERA. The reader is referred to Sections 3.0 through 3.5 for more details on the risk assessment. Conclusions regarding potential risks must be viewed in the context of the uncertainties associated with the assessment (Section 3.5) and the available risk information. The available risk information includes chemical data, exposure estimates, and literature-based toxicity information.

Table 3-24 summarizes the ecological risk estimates at the Cape Lisburne installation sites. The table shows the COCs that are associated with potential risk on a site-by-site basis for current and future conditions.

#### **3.6.1 Potential Risks to Representative Plants**

A qualitative comparison was conducted of onsite soil and surface water concentrations with plant toxicity information. The risk to plants is characterized using comparative information from the literature and BCF ( $B_v$ ). The risk to plants from PCBs at the locations that were sampled may be significant, although the risk is mitigated because the sample locations are poor habitat for plant growth (i.e., inside structures, on gravel pads). Based on the qualitative comparisons, the estimated risks to plants at the Cape Lisburne sites are not significant.

#### **3.6.2 Potential Risks to Representative Aquatic Species**

Potential risks to aquatic species (i.e., *Daphnia* spp.) were evaluated by comparing toxicity information from the literature with the average exposure concentrations of potential contaminants

**TABLE 3-24. SUMMARY OF ECOLOGICAL RISK ESTIMATES AT THE CAPE LISBURNE RADAR INSTALLATION SITES**

SITE	COC CONTRIBUTING TO RISK	RECEPTOR GROUPS AT RISK	CURRENT RISK POTENTIAL	FUTURE RISK POTENTIAL
Landfill and Waste Accumulation Area (LF01)	PCBs - Aroclor 1254 and Aroclor 1260	Birds, mammals	Low	Potential to increase over current estimate*
White Alice Site (SS03)	PCBs - Aroclor 1260	Birds, mammals	Low to moderate	Potential to increase over current estimate*
Spill/Leak #3 (ST07)	None	None	Not likely	Not likely
Upper Camp Transformer Building (SS08)	PCBs - Aroclor 1254 and Aroclor 1260	Birds, mammals	Low to moderate	Potential to increase over current estimate*
Lower Camp Transformer Buildings (SS09)	PCBs - Aroclor 1254 and Aroclor 1260	Birds, mammals	Low to moderate	Potential to increase over current estimate*

\* PCBs have a high potential for bioaccumulation and future PCB concentrations may be magnified in the food chain.

in surface water. HQs for *Daphnia* spp. indicate that risks may exist from iron and manganese; however, the use of total concentrations may overestimate the HQs. The dissolved concentrations of iron and manganese are within a range that is unlikely to cause adverse effects. Therefore, the overall risk to aquatic organisms at the Cape Lisburne sites is not estimated to be significant.

### 3.6.3 Potential Risks to Representative Species of Birds and Mammals

The risks to representative species of birds and mammals were evaluated using the quotient method. This method compares the estimated exposure with TRVs. The resulting HQs indicate that, with the exception of the risks posed by PCBs, the overall risks to birds and mammals are not significant. The PCB risks are estimated to range from low to moderate. In addition, there may be a potential for the PCB risk estimates to increase in the future as a result of bioaccumulation. Although HQs represent potential risks due to PCB exposure, limited access to the contaminated areas within the Upper Camp and Lower Camp Transformer Buildings lessens the potential for receptors to be exposed to PCBs.

The objective of this ERA was to evaluate the potential risk to the representative plant, aquatic, and terrestrial species at the five Cape Lisburne sites. This assessment indicates that risks posed by PCBs (Aroclor 1254 and Aroclor 1260) may be significant, currently and in the future, while the potential risk attributed to the other COCs is currently not significant and is likely to diminish in the future.

THIS PAGE INTENTIONALLY LEFT BLANK



#### 4.0 REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Manganese. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1991a. Toxicological Profile for 1,4-Dichlorobenzene. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1991b. Toxicological Profile for p-Cresol. Draft Report. U.S. Department of Health and Human Services.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1993. Toxicological Profile for Fuel Oils - Draft. U.S. Department of Health and Human Services. November 29, 1993.
- Alaska Biological Research. 1994. Spectacled and Steller's Eiders Surveys at 11CEOS Remote Sites in Alaska, 1994. Fairbanks, Alaska. 30 September 1994.
- Alaska Department of Environmental Conservation. 1991. Interim Guidance for Non-UST Contaminated Soil Cleanup Levels. Guidance Number 001, Revision 1. Alaska Department of Environmental Conservation, Juneau, Alaska. July 17, 1991.
- Alaska Department of Environmental Conservation. 1992. Comments on Cape Lisburne Long Range Radar Station Fuel Spill. Correspondence from Laura Noland of ADEC to Air Force. 17 November.
- Albers, P.H. 1977. Effects of External Applications of Fuel Oil on Hatchability of Mallard Eggs. Pages 158-163 in D.A. Wolfe (Ed). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. Pergamon Press, New York.
- Ambrose, S. 1994. Personal communication with S. Ambrose, a threatened and endangered species specialist with the U.S. Fish and Wildlife Service. Don Kellett, ICF Kaiser Engineers, Lakewood, Colorado. 31 March 1994.
- American Petroleum Institute (API). 1994. Results of Toxicological Studies Conducted for the American Petroleum Institute. API Publication No. 45591. January 1994.
- Aquatic Information Retrieval (AQUIRE). 1990. Computerized Database. Chemical Information System, Inc. Baltimore, Maryland.
- Aquatic Information Retrieval (AQUIRE). 1994. Computerized Database. Chemical Information System, Inc. Baltimore, Maryland.
- Armstrong Laboratory. 1994. Evaluation of the Total Petroleum Hydrocarbon Standard at Jet Fuel Contaminated Air Force Sites. Prepared by EA Engineering, Science and Technology Inc. for Armstrong Laboratory, Brook AFB, Texas. January.

- Baes, C.F., III, R.D. Sharp, A.L. Sjoreen, and R.W. Shor. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Oak Ridge National Laboratory. Prepared for the U.S. Department of Energy, Contract No. DE-AC05-84OR21400.
- Baudo, R., Giesy, J., and H. Muntau. 1990. Sediments: Chemistry and Toxicity of In-Place Pollutants. Lewis Publishers. Chelsea, Michigan.
- Beck, L.S., Hepler D.I., and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. Pages 1-12 in H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kaen (Eds). Proceedings of the Symposium - The Toxicology of Petroleum Hydrocarbons. American Petroleum Institute. Washington, D.C.
- Belopol'skii, L.O. 1961. Ecology of Sea Colony Birds of the Barents Sea. Translated from the Russian original. Israel Program for Scientific Translations. Jerusalem, Israel.
- Bergman, R.D., R.L. Howard, K.F. Abraham, and M. W. Weller. 1977. Water Birds and their Wetland Resources in Relation to Oil Development at Storkersen Point, Alaska. Resource Publication 129. U.S. Fish and Wildlife Service. Washington D.C.
- Beyer, N., E. Conner, and S. Gerould. 1994. Estimates of Soil Ingestion by Wildlife. J. Wildl. Manage. 58(2):375-382.
- Bott, T.L. and K. Rogenmuser. 1978. Effects of No. 2 Fuel Oil, Nigerian Crude Oil, and Used Crankcase Oil on Attached Algal Communities: Acute and Chronic Toxicity of Water-Soluble Constituents. Applied and Environmental Microbiology. November 1978:673-682.
- Brewster K. 1994. Personal communication with North Slope Borough oral historian.
- Burt, W.H. and R.P. Grossenheider (Eds). 1976. A Field Guide to the Mammals. Third Edition. Houghton-Mifflin Co., Boston, Massachusetts.
- Calder, W.A. and E.J. Braun. 1983. Scaling of Osmotic Regulation in Mammals and Birds. Am J. Physiol. 244:R601-R606.
- Cameron, R.D., D.J. Reed, J.R. Dau, and W.T. Smith. 1992. Redistribution of Calving Caribou in Response to Oil Field Development on the Arctic Slope of Alaska. Arctic 45(4):338-342.
- Chance, N. 1990. The Inupiat and Arctic Alaska: An Ethnography of Development. Holt, Rinehart, and Winston, New York. pp. 241.
- Chappell, M.A. 1980. Thermal Energetics and Thermoregulatory Costs of Small Arctic Mammals. J. Mamm. 61(2):278-291.
- Chesemore, D.L. 1967. Ecology of the Arctic Fox in Northern and Western Alaska. M.S. Thesis, University of Alaska, Fairbanks.

- Clayton, G.D. and F.E. Clayton (Eds). 1981-1982. *Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology*. Third Edition. New York: John Wiley Sons, 3292 pp.
- Cuccarese, S.V., M.F. Arend, R.J. Hensel, and P.O. McMillan. 1984. Biological and Socioeconomic Systems of the BAR-M, POW-1, LIZ-3A and SI-1 North Warning System Sites, Alaska. AEIDC. University of Alaska, Anchorage.
- Custance, S.R., P.A. McCaw, A.C. Kopf, and M.J. Sullivan. 1992. Environmental Fate of the Chemical Mixtures: Crude Oil, JP-5, Mineral Spirits and Diesel Fuel. *Journal of Soil Contamination* 1(4):379-386.
- Custer, T.W. and F.A. Pitelka. 1977. Demographic Features of a Lapland Longspur Population near Barrow, Alaska. *Auk* 94:505-525.
- Custer, T.W. and F.A. Pitelka. 1978. Seasonal Trends in Summer Diet of the Lapland Longspur near Barrow, Alaska. *Condor* 80:295-301.
- Custer, T.W. and G.H. Heinz. 1980. Reproductive Success and Nest Attentiveness of Mallard Ducks Fed Aroclor 1254. *Environ. Pollut.* 21:313-318.
- Derksen, D.V., T.C. Rothe, and W.D. Eldridge. 1981. Use of Wetland Habitats by Birds in the National Petroleum Reserve - Alaska. U.S. Fish and Wildlife Service, Resource Publication 141. Washington, D.C.
- Doudoroff, P. and M. Katz. 1953. Critical Review of Literature on the Toxicity of Industrial Wastes and their Components to Fish. II. The Metals. *Sewage and Industrial Wastes*. 25(7):802-839.
- Dunning, J.B. 1984. Body Weights of 686 Species of North American Birds. Western Bird Banding Association. Monograph No. 1. Cave Creek, Arizona.
- Eberhardt, L.E., W.C. Hanson, J.L. Bengtson, R.A. Garrott, and E.E. Hanson. 1982. Arctic Fox Home Range Characteristics in an Oil-Development Area. *J. Wildl. Manage.* 46(1):183-190.
- Eisler, R. 1986. Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biological Report 85(1.7). Laurel, Maryland.
- Farrand, J., Jr. (Ed). 1983. *The Audubon Society Master Guide to Birding, Volume 2*. Alfred A. Knopf, New York, New York. 398 pp.
- Foy, C.D., R.L. Chaney, and M.C. White. 1978. The Physiology of Metal Toxicity in Plants. *Ann. Rev. Physiol.* 29:511-566.

- Gaur, J.P. 1988. Toxicity of Some Oil Constituents to *Selenastrum Capricornutum*. *Hydrobiol.* 16(6):617-620. In: AQUIRE 1994.
- Geiger, J.G. and A.L. Bulkema, Jr. 1981. Oxygen Consumption and Filtering Rate of *Daphnia Pulex* after Exposure to Water-Soluble Fractions of Naphthalene, Phenanthrene, No. 2 Fuel Oil and Coal-Tar Creosote. *Bulletin of Environmental Contamination and Toxicology* 27:783-789.
- Harcharek, R.C. 1994. North Slope Borough 1993/94 Economic Profile and Census Report, Volume VII. North Slope Borough, Department of Planning and Community Services, Barrow, Alaska.
- Harding Lawson Associates. 1992. Offpost Operable Unit Endangerment Assessment/ Feasibility Study. Final Report. Technical support for Rocky Mountain Arsenal. 24 November 1992. Prepared for Program manager for Rocky Mountain Arsenal.
- Hart Crowser. 1987. Environmental Assessment for North Warning System. Alaska.
- Hartung, R. 1964. "Some Effects of Oils on Waterfowl." PhD Thesis. University of Michigan, Ann Arbor. In: R.C. Szaro, M.P. Dieter, G.H. Heinz, and J.F. Ferrell. 1978. Effects of Chronic Ingestion of South Louisiana Crude Oil on Mallard Ducklings. *Environmental Research* 17:426-436.
- Haseltine, S.D. and R.M. Prouty. 1980. Aroclor 1242 and Reproductive Successes of Adult Mallards (*Anas Platyrhynchos*). *Environ. Res.* 23:29-34).
- Hazardous Substance Data Bank (HSDB). 1994. National Institute of Health. Bethesda, Maryland.
- Heath, J.S., K. Koblis, and S.L. Sager. 1993. Review of Chemical, Physical, and Toxicologic Properties of Components of Total Petroleum Hydrocarbons. *J. of Soil Contamination.* 2(i): 1-25.
- Heath, R.G., J.W. Spann, E.F. Hill, and J.F. Kreitzer. 1972. Comparative Dietary Toxicities of Pesticides to Birds. U.S. Fish and Wildlife Service. Special Scientific Report - Wildlife 152. 57 pp.
- Hedtke, S. and F.A. Puglisi. 1982. Short-Term Toxicity of Five Oils to Four Freshwater Species. *Archives of Environmental Contamination and Toxicology* 11:425-430.
- Hensel, R., M.F. Arend, J. Thiele, P.O. McMilland, and S.V. Cuccarese. 1984. Living Resources of the Point Barrow, Oliktok Point, and Boulder Creek Areas, Alaska: A Literature Survey. AEIDC. University of Alaska, Anchorage.

- Hill, E.F. and M.B. Camardese. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to *Coturnix*. U.S. Fish and Wildlife Service. Technical Report 2. Washington, D.C. 147 p.
- Hoffman D.J. and W.C. Eastin. 1981. TOXICOL LETT 6: 35-40. In: HSDB 1994.
- Hudson, R.H., M.A. Haegele, and R.K. Tucker. 1979. Acute Oral and Percutaneous Toxicity of Pesticides to Mallards: Correlations with Mammalian Toxicity Data. Toxicology and Applied Pharmacology 47:451-460.
- Hull, R.N. and G.W. Suter II. 1994. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Sediment Associated Biota: 1994 Revision. ORNL Environmental Restoration Program. ES/ER/TM-95/R1.
- ICF Technology Incorporated (ICF). 1993. Work Plan for Remedial Investigations/Feasibility Studies at Air Force Radar Stations. Final Draft. Prepared for the U.S. Air Force.
- ICF Technology Incorporated. 1993. Literature Search, Pre-Survey, and Reconnaissance Visit to DEW Line and Cape Lisburne Air Force Station. Delivery Order 22.
- Integrated Risk Information System (IRIS). 1994. Environmental Criterion Assessment Office, U.S. Environmental Protection Agency, Cincinnati, Ohio.
- Integrated Risk Information System (IRIS). 1995. Integrated Risk Information System. Environmental Criterion Assessment Office, U.S. Environmental Protection Agency, Cincinnati, Ohio.
- Johnson, L. and B. Burns (Eds). 1984. Biology of the Arctic Char: Proceedings of the International Symposium on Arctic Char. University of Manitoba Press, Winnipeg, Man., Canada. 584 pp.
- Kabata-Pendias, A. and H. Pendias. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.
- Kauss, P.B. and T.C. Hutchinson. 1975. The Effects of Water-Soluble Petroleum Components on the Growth of *Chlorella Vulgaris* Beijerinck. Environmental Pollution (9):157-174.
- Klaassen, C.D., M.O. Amdur, and J. Doull. 1986. Casarett and Doull's Toxicology, the Basic Science of Poisons. Third Edition. MacMillan Publishing Company. New York. 974 pp.
- Kostecki, P.T. and E.J. Calabrese. 1989. Petroleum Contaminated Soils. Volume 1. Lewis Publishers, Chelsea, Michigan. 357 pp.
- Lewis, M.A., Evans, D.W., and J.G. Wiener. 1979. Manganese. In: R.V. Thurston, R.C. Russo, C.M. Ferrerolf, Jr., T.A. Edsall, and Y.M. Barber, Jr. (Eds.). 1979. A Review of the EPA

Red Book: Quality Criteria for Water. American Fisheries Society. Bethesda, Maryland. pp. 137-144.

Linzey, A.V. 1988. Effects of Chronic Polychlorinated Biphenyls Exposure on Growth and Reproduction of Second Generation White-Footed Mice (*Peromyscus Leucopus*). Arch. Environ. Contam. Toxicol. 17:39-45.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt (Eds). 1982. Research and Development Methods for Estimating Physio-chemical Properties of Organic Compounds of Environmental Concern. Report No. C-82426 by Arthur D. Little, Inc., under contract DAMD-17-78-C-0873, U.S. Army Medical R&D Command. Fort Detrick, Maryland.

Manahan, S.E. 1994. Environmental Chemistry. Sixth Edition. Lewis Publishers, Inc. Chelsea, Minnesota.

Mantel, N. and M.A. Schneiderman. 1975. Estimating Safe Levels: A Hazardous Undertaking. Cancer Res. 35:1379-1386.

Martin, A.C., H.S. Zim, and A.L. Nelson. 1961. American Wildlife and Plants: A Guide to Wildlife Food Habits. Dover Publications, New York. 500 pp.

Massachusetts Department of Environmental Protection (MDEP). 1993. Petroleum Policy: Development of Health-Based Alternative to the TPH Parameter. Prepared by ABB Environmental Services, Inc., Wakefield, Massachusetts. Project No. 06979-00. August.

MITRE. 1990. General Guidance for Ecological Risk Assessment at Air Force Installations. Prepared by the MITRE Corporation, Brooks Air Force Base, Texas. December 1990.

Nagy, K.A. 1987. Field Metabolic Rate and Food Requirement Scaling in Mammals and Birds. Ecol. Mono. 57:111-128.

National Academy of Sciences. 1980. Mineral Tolerance of Domestic Animals. Subcommittee on Mineral Toxicity in Animals. National Research Council. Washington, D.C.

National Oceanic and Atmospheric Administration (NOAA). August 1991. The Potential for Biological Effects of Sediment Sorbed Contaminants Tested in the National Status and Trends program. Technical Memorandum NOS OMA 52. NOAA, Seattle, Washington.

National Petroleum Reserve in Alaska Task Force (NPRA). 1978. Land Use Study, Volume 2. Values and Resource Analysis. U.S. Department of the Interior. Anchorage, Alaska.

Nowak, R.M. (Ed). 1991. Walker's Mammals of the World. Fifth Edition. Johns Hopkins University Press, Baltimore, Maryland.

O'Brien, J.W. 1978. Toxicity of Prudhoe Bay Crude Oil to Alaskan Arctic Zooplankton. Arctic 31(3):219-228.

- Opresko, D.M., B.E. Sample, and G.W. Suter II. 1994. Toxicological Benchmarks for Wildlife: 1994 Revision. ORNL Environmental Restoration Program. ES/ER/TM-86/R1.
- Palmer, R.S. (Ed). 1976. Handbook of North American Birds, Volume 2, pp. 244-273. Yale University Press, New Haven, Connecticut. 521 pp.
- Pitelka, F.A. 1959. Numbers, Breeding Schedule, and Territoriality in Pectoral Sandpipers of Northern Alaska. Condor 62(4):233-264.
- Platanow, N.S. and L.H. Karstad. 1973. Dietary Effects of Polychlorinated Biphenyls on Mink. Can. J. Comp. Med. 37:391-400.
- Puls, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.
- Raven, P.H., R.H. Evert, and F.E. Eichorn. 1986. Biology of Plants. Worth Publishers, Inc. New York, New York. 775 pp.
- Ringer, R.K. 1983. Toxicology of PCBs in Minks and Ferrets In: F.M. D'Itri and M.A. Kamrin (Eds.). PCBs: Human and Environmental Hazards. Ann Arbor Science Book. Butterworth Publishers, Boston, Massachusetts.
- Sax, I. and R.J. Lewis, Sr. 1989. Dangerous Properties of Industrial Materials. Seventh Edition. Van Nostrand, Reinhold, New York.
- Scott, S.L. 1983. Field Guide to the Birds. National Geographic Society. Washington, D.C. 464 pp.
- Skogland, T. 1980. Comparative Summer Feeding Strategies of Arctic and Alpine Rangifer. Journal of Animal Ecology. 49:81-98.
- Smith, G.J. 1987. Pesticide Use and Toxicology in Relation to Wildlife: Organophosphorus and Carbamate Compounds. U.S. Fish and Wildlife Service, Resource Publication 170. Washington, D.C.
- Snyder-Conn, E. 1994. Personal communication with E. Snyder-Conn, wildlife biologist with the U.S. Fish and Wildlife Service, Fairbanks, Alaska. Don Kellett, ICF Kaiser Engineers. 31 March 1994.
- Soto, C., J. Hellebust, and T.C. Hutchinson. 1975. Effect of Napthalene and Aqueous Crude Oil Extracts on the Green Flagellate *Chlamydomonas Angulosa*. Canadian J. of Botany 53(2):118-126.
- Spacie, A. and J.L. Hamelink. 1985. Bioaccumulation, Chapter 17. Pages 495-525 in G.M. Rand and S.R. Petrocelli (Eds). Fundamentals of Aquatic Toxicology. Hemisphere Publishing Corporation, New York, New York.

- Suter, G.W and J.B. Mabrey. 1994. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision. Environmental Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- Szaro, R.C. and P.H. Albers. 1977. Effects of External Applications of No. 2 Fuel Oil on Common Eider Eggs. Pages 164-167 in D.A. Wolfe (Ed). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. Pergamon Press, New York.
- Szaro, R.C., M.P. Dieter, G.H. Heinz, and J.F. Ferrell. 1978. Effects of Chronic Ingestion of South Louisiana Crude Oil on Mallard Ducklings. Environmental Research 17:426-436.
- Talmadge, S.S. and B.T. Walton. 1991. Small Mammals as Monitors of Environmental Contaminants. Reviews of Environ. Contam. Toxicol. 119:47-145.
- Travis, C.C. and A.D. Arms. 1988. Bioconcentration of Organics in Beef, Milk, and Vegetation. Environ. Sci. Technol. 22(3):271-274.
- Tucker, R.K. and J.S. Leitzke. 1979. Comparative Toxicology of Insecticides for Vertebrate Wildlife and Fish. Pharmac. Ter. 6:167-220.
- U.S. Air Force. 1991. Handbook to Support the Installation Restoration Program (IRP) Statements of Work. Volume 1, Remedial Investigation/Feasibility Studies (RI/FS). Human Systems Division, Brooks Air Force Base, Texas.
- U.S. Air Force. 1993. Sampling and Analysis Plan for DEW Line and Cape Lisburne Radar Stations. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Inc.
- U.S. Air Force. 1995. Final Interim Remedial Action Report for the Cape Lisburne Radar Station. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Inc.
- U.S. Air Force. 1996. Final Remedial Investigation/Feasibility Study for Cape Lisburne Radar Installation, Alaska. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Inc.
- U.S. Army Corps of Engineers (USACOE). 1991. Baseline Risk Assessment for Eight Selected Study Areas at Aberdeen Proving Ground. Appendix C, Draft Document.
- U.S. Environmental Protection Agency. 1976. Quality Criteria for Water. Office of Water and Hazardous Materials. Washington, D.C.
- U.S. Environmental Protection Agency. 1985. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Iron. Office of Water Regulations and Standards. Washington, D.C.



- U.S. Environmental Protection Agency. 1986a. Guidelines for Carcinogen Risk Assessment. Federal Register 51:33992-34013.
- U.S. Environmental Protection Agency. 1986b. Guidelines for Health Risk Assessment of Chemical Mixtures. Federal Register 51:34014-34025.
- U.S. Environmental Protection Agency. 1986c. Quality Criteria for Water. Office of Water Regulations and Standards. Washington D.C. EPA 440/5-86-001.
- U.S. Environmental Protection Agency. 1989a. Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A. Office of Solid Waste and Emergency Response. Washington D.C.
- U.S. Environmental Protection Agency. 1989b. Risk Assessment Guidance for Superfund: Volume 2, Environmental Evaluation Manual. Office of Solid Waste and Emergency Response. Washington D.C.
- U.S. Environmental Protection Agency. 1991a. Region 10 Supplemental Risk Assessment Guidance for Superfund. Seattle, Washington. 16 August 1991.
- U.S. Environmental Protection Agency. 1991b. Human Health Evaluation Manual, Part B: Development of Risk-based Preliminary Remediation Goals. Office of Solid Waste and Emergency Response. Washington D.C. December 13, 1991.
- U.S. Environmental Protection Agency. 1991c. Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. Office of Solid Waste and Emergency Response. Washington D.C. 22 April 1991.
- U.S. Environmental Protection Agency. 1992a. Framework for Ecological Risk Assessment. EPA/630/R-92/001. NTIS #PB93-102192. Washington D.C. February 1992.
- U.S. Environmental Protection Agency. 1992b. Oral Reference Doses and Oral slope Factors for JP-4, JP-5, Diesel Fuel, and Gasoline. Environmental Criterion Assessment Office, Office of Research and Development. Cincinnati, Ohio. March 24, 1992.
- U.S. Environmental Protection Agency. 1992c. Handbook of RCRA Ground-Water Monitoring Constituents: Chemical and Physical Properties. Office of Solid Waste Permits and State Programs Division. September 1992.
- U.S. Environmental Protection Agency. 1992d. Superfund Health Risk Technical Support Center. Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- U.S. Environmental Protection Agency (EPA). 1994. Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments. USEPA Environmental Response Team, Edison, New Jersey. September 26, 1994. Review Draft.

- U.S. Fish and Wildlife Service (USFWS). 1982. Arctic National Wildlife Refuge Coastal Plain Resource Assessment-Initial Report. Baseline Study of Fish, Wildlife and their Habitats. U.S. Department of the Interior, Anchorage Alaska.
- U.S. Fish and Wildlife Service (USFWS). 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to *Coturnix*. Fish and Wildlife Technical Report 2. Washington, D.C.
- University of Alaska, Arctic Environmental Information and Data Center. 1978. Kaktovik, pp 2-29.
- Verschueren, K. 1983. Handbook of Environmental Data of Organic Chemicals. Second Edition. New York: Van Nostrand Reinhold Co. P. 629
- Von Burg, R. 1993. Evaluation of TPH as a Determinant for Petroleum Hydrocarbon Cleanup in Soil. ICF Kaiser Engineers, Oakland, California.
- Walker, D.A., P.J. Webber, K.R. Everett, and J. Brown. 1978. Effects of Crude and Diesel Oil Spills on Plant Communities at Prudhoe Bay, Alaska, and the Derivation of Oil Spill Sensitivity Maps. Arctic 31(3):242-259.
- Weeks, J.A., G.H. Drendel, R.S. Jagan, T.E. McManus, and P.J. Sczerzenie. 1988. Diesel Oil and Kerosene Background Statement. Prepared by Labat-Anderson, Inc. for the U.S. Department of Agriculture, Forest Service. February.
- White, R.G. and J. Trudell. 1980. Habitat Preference and Forage Consumption by Reindeer and Caribou near Atkasook, Alaska. Arctic and Alpine Research 12(4):511-529.
- Will, M.E. and G.W. Suter. 1994. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision. Oak Ridge National Laboratory. Oak Ridge, Tennessee.
- Wong, C.K., F.R. Engelhardt, and J.R. Strickler. 1981. Survival and Fecundity of *Daphnia Pulex* on Exposure to Particulate Oil.
- Woodward-Clyde Corporation. 1993. Natural Resources Plan: North Coastal Long Range Radar Sites. Final Draft. Prepared for the United States Air Force.
- Wootton, R.J. 1976. The Biology of the Sticklebacks. Academic Press, New York. 387 pp.

**APPENDIX A**

**RISK CHARACTERIZATION SPREADSHEETS**

Landfill and Waste Accumulation Area (LF01) .....	A-1
White Alice Site (SS03) .....	A-5
Spill/Leak #3 .....	A-7
Upper Camp Transformer Building (SS08) .....	A-9
Lower Camp Transformer Building (SS09) .....	A-11

TABLE A-1. LANDFILL AND WASTE ACCUMULATION AREA (LF01) RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion  
 Endpoint: Noncancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Landfill and Waste Accumulation Area (LF01)  
 File: LF01SONC.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
DRPH	0.08	18,600	1.09e-03	2.18e-03	2.04e-02	1.36e-02	2.82e-01
RRPH	0.08	43,100	2.53e-03	5.06e-03	4.72e-02	3.16e-02	6.54e-01
Aroclor 1260	0.00002	999	5.86e-05	1.17e-04	1.09e-03	2.93e+00	6.06e+01
Carbon tetrachloride	0.0007	17.3	1.02e-06	2.03e-06	1.90e-05	1.45e-03	3.00e-02
HAZARD INDEX							61.570

**TABLE A-2. LANDFILL AND WASTE ACCUMULATION AREA (LF01) RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Cancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Landfill and Waste Accumulation Area (LF01)  
 File: LF01SOCA.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
Carbon tetrachloride	0.13	17.3	1.45e-07	1.42e-06	1.63e-06	1.89e-08	3.96e-07
Trichloroethene	0.011	15.3	1.28e-07	1.28e-06	1.44e-06	1.41e-09	2.96e-08
Aroclor 1260	7.7	999	8.38e-06	8.21e-05	9.38e-05	6.45e-05	1.35e-03
CANCER RISK							1e-03

**TABLE A-3. LANDFILL AND WASTE ACCUMULATION AREA (LF01) RISK ASSESSMENT SPREADSHEET**

Route: Water Ingestion  
 Endpoint: Noncancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Landfill and Waste Accumulation Area (LF01)  
 File: LF01WANC.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	180	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(ED x 365 days/year)	3,650	20,075	NA

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)			Hazard Quotient	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult
Carbon tetrachloride	0.0007	0.089	1.25e-03	1.25e-03	NA	1.79e+00	1.79e+00
Tetrachloroethene	0.01	0.004	5.64e-05	5.64e-05	NA	5.64e-03	5.64e-03
Chloroform	0.01	0.016	2.25e-04	2.25e-04	NA	2.25e-02	2.25e-02
Barium	0.07	0.86	2.00e-03	1.20e-03	NA	2.90e-02	1.73e-01
Manganese	0.005	1.8	2.54e-02	2.54e-02	NA	5.07e+00	5.07e+00
HAZARD INDEX							6.89e+00

**TABLE A-4. LANDFILL AND WASTE ACCUMULATION AREA (LF01) RISK ASSESSMENT SPREADSHEET**

Route: Water Ingestion  
 Endpoint: Cancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Landfill and Waste Accumulation Area (LF01)  
 File: LF01WACA.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	180	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(lifetime in days)	25,550	25,550	NA

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult
Benzene	0.029	0.005	1.01e-05	5.54e-05	NA	2.92e-07	1.61e-06
Tetrachloroethene	0.052	0.004	8.05e-06	4.43e-05	NA	4.19e-07	2.30e-06
Carbon tetrachloride	0.13	0.089	1.79e-04	9.85e-04	NA	2.33e-05	1.28e-04
Trichloroethene	0.011	0.021	4.23e-05	2.32e-04	NA	4.65e-07	2.56e-06
Chloroform	0.0061	0.016	3.22e-05	1.77e-04	NA	1.96e-07	1.08e-06
CANCER RISK							2e-05
							1e-06

**TABLE A-5. WHITE ALICE SITE (SS03) NSTALLATION RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Noncancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: White Alice Site (SS03)  
 File: SS03SONC.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
Aroclor 1260	0.00002	6,290	3.69e-04	7.39e-04	6.89e-03	1.85e+01	3.82e+02
HAZARD INDEX							381.585



**TABLE A-6. WHITE ALICE SITE (SS03) RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Cancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: White Alice Site (SS03)  
 File: SS03SOCA.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
Aroclor 1260	7.7	6,290	5.28e-05	5.17e-04	5.91e-04	4.06e-04	8.53e-03
			CANCER RISK				
						4e-04	9e-03

**TABLE A-7. SPILL/LEAK #3 (ST07) RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Noncancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Spill/Leak #3 (ST07)  
 File: ST07SONC.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
DRPH	0.08	63,000	3.70e-03	7.40e-03	6.90e-02	4.62e-02	9.55e-01
GRPH	0.2	150	8.81e-06	1.76e-05	1.64e-04	4.40e-05	9.10e-04
HAZARD INDEX							0.956

**TABLE A-8. SPILL/LEAK #3 (ST07) RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Cancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Spill/Leak #3 (ST07)  
 File: ST07SOCA.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
GRPH	0.0017	150	1.26e-06	1.23e-05	1.41e-05	2.14e-09	4.49e-08
Benzene	0.029	1.4	1.17e-08	1.15e-07	1.32e-07	3.41e-10	7.15e-09
CANCER RISK							5e-08

TABLE A-9. UPPER CAMP TRANSFORMER BUILDING (SS08) RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion  
 Endpoint: Noncancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Upper Camp Transformer Building (SS08)  
 File: SS08SONC.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
DRPH	0.08	51,000	2.99e-03	5.99e-03	5.59e-02	3.74e-02	7.73e-01
RRPH	0.08	29,000	1.70e-03	3.41e-03	3.18e-02	2.13e-02	4.40e-01
Aroclor 1254	0.00002	29.1	1.71e-06	3.42e-06	3.19e-05	8.54e-02	1.77e+00
Aroclor 1260	0.00002	300	1.76e-05	3.52e-05	3.29e-04	8.81e-01	1.82e+01
HAZARD INDEX						1.025	21.178

**TABLE A-10. UPPER CAMP TRANSFORMER BUILDING (SS08) RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Cancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Upper Camp Transformer Building (SS08)  
 File: SS08SOCA.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
Aroclor 1254	7.7	29.1	2.44e-07	2.39e-06	2.73e-06	1.88e-06	3.95e-05
Aroclor 1260	7.7	300	2.52e-06	2.47e-05	2.82e-05	1.94e-05	4.07e-04
CANCER RISK							4e-04

TABLE A-11. LOWER CAMP TRANSFORMER BUILDINGS (SS09) RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion  
 Endpoint: Noncancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Lower Camp Transformer Buildings (SS09)  
 File: SS09SONC.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
DRPH	0.08	30,000	1.76e-03	3.52e-03	3.29e-02	2.20e-02	4.55e-01
RRPH	0.08	13,000	7.63e-04	1.53e-03	1.42e-02	9.54e-03	1.97e-01
Aroclor 1254	0.00002	1,720	1.01e-04	2.02e-04	1.88e-03	5.05e+00	1.04e+02
Aroclor 1260	0.00002	5,600	3.29e-04	6.58e-04	6.14e-03	1.64e+01	3.40e+02
HAZARD INDEX						21.519	444.723

**TABLE A-12. LOWER CAMP TRANSFORMER BUILDINGS (SS09) RISK ASSESSMENT SPREADSHEET**

Route: Soil Ingestion  
 Endpoint: Cancer  
 Assumptions: Site-specific  
 Installation: Cape Lisburne  
 Site: Lower Camp Transformer Buildings (SS09)  
 File: SS09SOCA.WK1

Exposure Assumptions		Installation Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	30	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	1.00e-06	1.00e-06	1.00e-06
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			Installation Worker	Native Northern Adult	Native Northern Child	Installation Worker	Native Northern Adult/Child
Aroclor 1254	7.7	1,720	1.44e-05	1.41e-04	1.62e-04	1.11e-04	2.33e-03
Aroclor 1260	7.7	5,600	4.70e-05	4.60e-04	5.26e-04	3.62e-04	7.59e-03
CANCER RISK							1e-02

## **APPENDIX B**

### **TOXICITY PROFILES**

DIESEL FUEL (DRPH) .....	B-1
GASOLINE (GRPH) .....	B-6
WASTE OIL (RRPH) .....	B-11
TETRACHLOROETHENE .....	B-17
BARIUM .....	B-19
MANGANESE .....	B-22
BENZENE .....	B-25
TRICHLOROETHENE .....	B-27
AROCOR 1254 (POLYCHLORINATED BIPHENYLS, PCBs) .....	B-29
CARBON TETRACHLORIDE .....	B-32
CHLOROFORM .....	B-34



## TOXICOLOGY PROFILE FOR DIESEL FUEL (DRPH)

### GENERAL DATA

Petroleum fuels are classified into light, middle, and heavy distillate fuels. Gasoline is a typical light distillate fuel while diesel fuel is considered to be a middle distillate material obtained from the distillation of crude oil. Included in this category of middle distillate fuels are jet fuel, kerosene, and #2 fuel oils. Many of the ecological and toxicological effects of these materials are very similar.

The chemical composition of diesel fuel is extremely variable and depends upon the crude oil source, types of processing and refining, blending, and additives employed. These fuels are formulated to meet physical characteristics and not a specific chemical composition. Viscosity and volatility are the principal determinants of the fuel specifications. Diesel #1 is primarily a kerosene type of fuel and produced mainly from straight run middle distillates. Diesel #2 also contains straight run middle distillate but is also blended with straight run kerosene, straight run gas oils, light vacuum distillate, and light thermally and/or catalytically cracked streams (IARC 1989).

Like other petroleum derived fuels, diesel fuels consist of paraffins, olefins, cycloparaffins, isoparaffins, and aromatics as well as additives. Additives can include amyl nitrates, alcohols, n-hexyl nitrate, and octyl nitrate at levels of 0.1 - 0.2 percent (Kirk-Othmer 1984). The total aromatic content of diesel fuel is also variable but levels between 23 - 38 percent have been reported. The average total aromatic is probably in the range of 25 percent. The concentrations of the principal aromatic species of toxicological significance is presented in Table B-1.

### FATE AND TRANSPORT

Microbial degradation, plus evaporation, can remove up to 90 percent of the added diesel fuel to soil. Depending on the soil characteristics, the half-life of diesel fuel in soil ranges for one to eight weeks (Song 1988). Volatilization to the air occurs and diesel fuel can be detected by its odor in the air. However, a vapor pressure value could not be located in the literature. Diesel fuel will percolate through the soil and float on the ground water. When spilled onto surface water, diesel fuels can be toxic to fish, waterfowl and algae.

**TABLE B-1. REPRESENTATIVE VALUES FOR TOXICOLOGICALLY SIGNIFICANT AROMATIC CONTENT FOR DIESEL FUEL# 2.**

COMPONENT	APPROXIMATE CONCENTRATION
Benzene	<50 ppm with an average of 10 ppm
Ethylbenzene	300 ppm
Toluene	200 ppm (max)
Xylene (mixed)	2,400 ppm

(personal communication, Chevron Corp.)

The odor threshold of diesel fuel is approximately 0.8 ppm.

## **TOXICITY DATA**

### **Human Toxicological Profile**

Like other solvents, diesel fuel can be expected to be a central nervous system (CNS) depressant. However, since this fuel is not as volatile as gasoline, breathing vapors at concentrations sufficient to achieve a level of intoxication is not likely at normal temperatures and pressures. An attempt to generate a kerosene (diesel) laden atmosphere only resulted in an ambient concentration of 14 ppm (Carpenter et al. 1976). However, under certain occupational settings like tank cleaning, it may be possible to generate mists or aerosols that can lead to symptoms of overexposure. As with kerosene, these symptoms may include headache, dizziness, weakness, confusion, drowsiness and possibly death (HSDB 1991).

Ingestion of diesel fuel can occur during siphoning, abuse situations, or from contaminated well-water. Ingestion may be accompanied by a burning sensation in the mouth, pharynx and chest, gastrointestinal hypermotility and diarrhea (Gosselin et al. 1984), and possibly nausea and vomiting. A serious complication is the aspiration of hydrocarbons into the lung which produces a potentially-lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

There have been reports of acute renal failure following persons exposed to diesel fuel (Barrientos et al. 1977; Crisp et al. 1979). Kryzanovskij (1971) reports that workers cleaning diesel storage tanks have an increased incidence of disease in general, and specifically cardiovascular disease and bronchitis over control shipyard workers.

### **Animal Toxicology and Significant Studies**

The acute oral and dermal LD<sub>50</sub> of diesel fuel is in the range of 9 ml/kg body weight. Eye irritation properties were minimal, but the primary skin irritation score of a marketplace sample was 6.8 indicating that this material is a strong skin irritant (Beck et al. 1982). Chronic skin contact can be expected to produce defatting, fissuring and cracking. There are no readily available reports on hypersensitivity response to diesel fuels can be expected to occur since products on either side of diesel fuels distillation range have been reported to produce hypersensitivity reactions (Beck et al. 1982). Dermal absorption of gasoline is unlikely to result in systemic toxicity, but chronic poisoning of the readily absorbable alkyl lead additives is possible.

Exposure of CD-1 mice to diesel vapor for eight hours per day on five consecutive days resulted in a decrement of performance on the roto-rod test, square box activity test and hot plate test. However, the corneal reflex and inclined plane test was unaffected. General observations noted vasodilation, ataxia, poor grooming and in some cases tremor (Kainz and White 1982).

Exposure of rats to aerosolized diesel fuel at concentrations up to 6 mg/L produced direct toxic effects on the lungs but did not produce any neurotoxicity (Dalbey et al. 1987).

### **Reproductive Toxicity**

Female rats were exposed 6 hours per day to air concentrations of 0, 100, and 400 ppm during days 6 through 15 of gestation. Neither jet fuel or No. 2 Fuel Oil produced any significant detrimental effects on the reproductive parameters of the experimental animals (Beliles and

Mecler 1982). Neither Jet Fuel A or diesel fuel at exposure levels of 400 ppm, 6 hrs per day, 5 day per week for 8 weeks reduced the fertility of CD-1 male mice (API 1980a,b).

### **Genotoxicity**

Kerosene, jet fuel and diesel fuel all tested negatively in the standard Ames bioassay. However, the "Modified Ames Assay" (Blackburn et al. 1988) on two straight run gas oils did demonstrate mutagenicity. (Straight run gas oil can be considered similar to diesel oils.) Diesel fuel was also negative in the mouse lymphoma assay but positive on the rat bone marrow cytogenetics assay when administered by intraperitoneal injection (Conaway et al. 1982). Heating oil #2 produced a positive Ames test as well as positive results in two other short term bioassays (Rothman and Emmett 1988). Dominant lethal testing of Jet fuel A and diesel fuel was negative at 400 ppm to male CD-1 mice (API 1980a,b).

### **Carcinogenicity**

In a classical mouse skin painting bioassay, all petroleum fractions derived from a crude oil source that boiled between 120 and 700°F showed a low level of tumorigenic activity (Lewis et al. 1982). Home heating oil also showed a low level of tumorigenicity in a more recent mouse skin painting assay (Witschi et al. 1987).

In a case referent study, Seimietycki et al. (1987) reported an increase of several specific cancers associated with exposures to different petroleum products. Leaded gasoline was associated with stomach cancer; aviation gasoline with kidney cancer; diesel fuel with non adenocarcinoma of the lung and prostate cancer and mineral spirits with squamous cell lung cancer. However not all parameters of concern were properly controlled, excluded or assessed making conclusions from this study inappropriate.

IARC (1989) has classified diesel fuel as having limited evidence of carcinogenicity in animals. Light diesel fuels are not classifiable as to their carcinogenicity to humans (Group 3).

### **REGULATIONS AND STANDARDS**

Neither the American Conference of Governmental Industrial Hygienists (ACGIH) nor OSHA have recommended or established permissible exposure standards (PELs) for diesel fuels. However, NIOSH, has recommended a 10 hour time-weighted average of 100 mg/m<sup>3</sup> or 14 ppm for kerosene (NIOSH 1977). Because of the complexity and variability in composition, OSHA regulates the toxic components by their respective PELs (i.e., n-hexane, benzene, etc.).

Diesel fuels, as such, are not mentioned in HEAST (1990) nor identified for a specific cancer Potency Factor (CPF) or reference dose (RfD). However, individual components such as benzene, other aromatics and for n-hexane having CPF or RfD values should be evaluated by themselves.

### **REFERENCES**

- API. 1980a. Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-03. American Petroleum Institute Medical Research Publications, Washington, D.C.

- API. 1980b. Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-04. American Petroleum Institute Medical Research Publications, Washington, D.C.
- Barrientos, A., M.T. Ortuno, and J.M. Morales et al. 1977. Acute Renal Failure After Use of Diesel Fuel as a Shampoo. Arch.Intern. Med. 137:1217-1219.
- Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Beliles, R.P. and F.J. Mecler. 1982. Inhalation Teratology of Jet Fuel, Fuel Oil and Petroleum Naphtha in Rats. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Blackburn, G.R., R.A. Deitch, and T.A. Roy et al. 1988. Estimation of the Dermal Carcinogenic Potency of Petroleum Fractions Using a Modified Ames Assay. In: Polynuclear Aromatic Hydrocarbons: A Decade of Progress, Proceedings of the Tenth International Symposium. M. Cooke and A.J. Dennis, editors. Battelle Press, Columbus Ohio, 99 83-97.
- Carpenter, C.P., D.L. Geary, and R.C. Meyers et al. 1976. Petroleum Hydrocarbon Toxicity Studies XI. Animal and Human Response to Vapors of Deodorized Kerosene. Toxicol. Appl. Pharmacol. 36:443-456.
- Conaway, C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity Evaluation of Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Crisp, A.J., A.K. Bhalla, and B.I. Hoffbrand. 1979. Acute Tubular Necrosis After Exposure to Diesel Oil. Brit. Med J. 2:177-178.
- Dalbey, W., M. Henry, and R. Holmberg et al. 1987. Role of Exposure Parameters in the Toxicity of Aerosolized Diesel Fuel in the Rat. J. Appl. Toxicol. 7:265-275.
- Gosselin, R.E., R.P. Smith, and H.C. Hodge. 1984. Clinical Toxicology of Commercial Products. 5th Ed. Baltimore, Maryland: Williams & Wilkins.
- HEAST. 1990. Health Effects Assessment Summary Tables, Office of Emergency and Remedial Response, Environmental Protection Agency. OERR 9200 6-303, (90-4).
- HSDB. 1991. Hazardous Substances Data Bank. National Library of Medicine. Washington, D.C.
- IARC. 1989. Monographs on the Evaluation of Carcinogenic Risks to Humans. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. International Agency for Research on Cancer, Lyon France. Vol 45.

- Kainz, R.J. and L.E. White. 1982. Consequences Associated with the Inhalation of Uncombusted Diesel Vapor. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Kirk Othmer. 1984. Diesel Fuel. In: Kirk-Othmer Encyclopedia of Chemical Technology. F. Mark, M. Grayson, and D. Eckroth et al., Eds. 3rd Ed. John Wiley and Sons, New York.
- Kryzanovskij, N.V. 1971. Occupational Health Conditions Associated with the Cleaning of Oil Tankers and Their Effect on Worker's Health. Gig. Tr. Prof. Zabol. 15:14-17.
- Lee T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. (Letter). The Lancet 8134:149.
- Lewis, S.C., R.W. King, S.T. Cragg, and D.W. Hillman. 1982. Skin Carcinogenic Potential of Petroleum Hydrocarbons. 2. Carcinogenesis of Crude Oil, Distillate Fractions and Chemical Class Subfractions. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- NIOSH. 1977. Criteria for a Recommended Standard...Occupational Exposure to Refined Petroleum Solvents. National Institute for Occupational Safety and Health, Cincinnati, Ohio. NIOSH Publication #77-192.
- Rothman, N. and E.A. Emmett. 1988. The Carcinogenic Potential of Selected Petroleum-derived Products. Occup. Med.: State Art Rev. 3:475-482.
- Siemiatycki, J., R. Dewar, and L. Nadon et al. 1987. Associations Between Several Sites of Cancer and Twelve Petroleum Derived Liquids. Scan. J. Work Environ. Health. 13:493-504.
- Song, H.G. 1988. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey.
- Witschi, H.P., L.H. Smith, and E.L. Frome et al. 1987. Skin Tumorigenic Potential of Crude and Refined Coal Liquids and Analogous Petroleum Products. Fund. Appl. Toxicol. 9:297-303.

## **TOXICOLOGY PROFILE FOR GASOLINE (GRPH)**

### **GENERAL DATA**

The chemical composition of gasoline is extremely variable, depending upon the crude oil starting material, types of processing and refining, blending and additives employed. Gasolines are formulated to meet fuel performance specifications, not to achieve a specific chemical composition. Volatility must be within a certain range to avoid vapor lock (too high) or sluggish acceleration (too low). In addition, the air-fuel mixture within the cylinder must burn uniformly to prevent "pinging" or "knocking." Often small quantities of butanes, pentanes, organo lead compounds or branched chain hydrocarbons are added to achieve uniform burning rates. McDermott and Killiany (1978) published a detailed gas chromatographic analysis of a premium grade gasoline listing 21 components which accounted for 92 percent of the gasoline vapors (Table B-2). Low-volatility hydrocarbons (high carbon numbers) were not well represented.

Gasoline additives include organic lead (tetraethyl lead and tetramethyl lead) to a concentration of 0.1 g/gallon (7 ppm). Alkyl lead vapors have low volatility (vapor pressure = 0.4 mm Hg) compared to gasoline (400-775 mm Hg), so lead compounds should not be acutely hazardous by inhalation. To prevent accumulation of lead deposits, scavenging agents are added to fuels: ethylene dichloride (EDC) and ethylene dibromide (EDB), usually in a molar ratio EDC/EDB/Pb = 2:2:1.

### **FATE AND TRANSPORT**

Gasoline released into the environment would be expected to evaporate rapidly due to its high vapor pressure (400-477 mm Hg). Studies of gasoline fate when added to soils show that the main clearance mechanism was evaporation which can account for up to 75 percent removal from surface soils (Donaldson 1990). Microbial degradation, plus evaporation, can remove up to 90 percent of the added gasoline (Song 1988). Benzene, a volatile gasoline component of major toxicological interest, has a half life in the air of less than one day (Korte and Klein 1982). Gasoline has appreciable water solubility (12-16 percent) so it would be transported in ground water and may be found in well water.

### **TOXICITY DATA**

#### **Human Toxicological Profile**

Like other solvents, gasoline has potent central nervous system (CNS) depressant activity. Breathing vapors at concentrations achieved during "huffing" or occupational overexposures has led to a variety of neurological symptoms: hallucinations, encephalopathy, ataxia, convulsions, Tourette's Disease, vertigo and nystagmus and peripheral neuropathy (Von Burg 1989). Many of these symptoms may be attributed to n-hexane or alkyl lead compounds.

Ingestion of gasoline can occur during siphoning, abuse situations, or from contaminated well-water. Ingestion is accompanied by a burning sensation in the mouth, pharynx and chest. Swallowing large amounts of gasoline leads to coma and death by respiratory depression. A serious complication is the aspiration of hydrocarbons into the lung which produces a potentially-lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

**TABLE B-2. COMPOSITION OF A PREMIUM-GRADE GASOLINE**

COMPOUND	VOL %
Propane	0.8
n-Butane	38.1
Isobutane	5.2
n-Pentane	7.0
Cyclopentane	0.7
2,3-DM-butane	0.7
2-M-pentane	2.1
3-M-pentane	1.6
n-Hexane	1.5
M-cyclopentane	1.3
2,4-DM-pentane	0.4
2,3-DM-pentane	0.7
2,2,4-TM-pentane	0.5
Isobutylene	1.1
2-M-1-butane	1.6
c-2-pentene	1.2
2-M-2-butene	1.7
Benzene	0.7
Toluene	1.8
Xylene (m,p,o)	0.5
Total %	92.1

Inhalation exposure to gasoline at concentrations of 1,000 - 5,000 ppm for 15 - 60 minutes can produce CNS depression. A 5-minute exposure to 20,000 ppm (20 percent) has been reported to be fatal (Von Burg 1989).

Three epidemiologic studies of refinery workers showed no increased cancer risk in refinery workers (Hanis et al. 1982; Kaplan 1986; Wong 1987). In an epidemiological study of refinery workers and gasoline handlers, Thomas et al. (1982) found a significant increase in stomach and brain cancer with a trend to increased leukemia and cancer of the skin, prostate and pancreas.

### **Animal Toxicology and Significant Studies**

The acute dermal LD<sub>50</sub> of gasoline in rabbits is reported to be <5 ml/kg (Von Burg 1989). Liquid gasoline is considered a primary skin irritant because of the defatting and fissuring which occurs upon repeated contact. Hypersensitivity response to gasoline can occur. Dermal absorption of gasoline is unlikely to result in systemic toxicity, but chronic poisoning of the readily absorbable alkyl lead additives is possible. Gasoline is acutely irritating to the eye, animal studies indicate no effect lasting longer than seven days.

MacFarland (1982), reported on a chronic inhalation study of gasoline in Fischer 344 rats and B6C3F<sub>1</sub> mice. Exposure levels were 0, 67, 292, and 2,056 ppm for 6 hours/day, 5 days/week for 103 to 113 weeks. Male (but not female) rats exhibited a progressive renal tubular disease and renal carcinomas in all dose groups; renal effects in mice were within the expected range of control. High dose female mice had an increased incidence of hepatocellular tumors (48 percent), but the spontaneous incidence of these tumors is also high (14 percent); males showed no increase (44 percent high dose vs. control 45 percent).

### **Reproductive Toxicity**

Male rats exposed intermittently to about 650 ppm unleaded petrol for 2 months showed endocrine changes which were attributed to stress. The offspring of pregnant females exposed to 0, 400, and 1,600 ppm unleaded gasoline for 6 hours per day on days 6-15 of gestation did not show any teratogenic or fetotoxic effects. Mental retardation has been reported among the offspring of gasoline-sniffing mothers.

### **Genotoxicity**

Negative results were observed with several common fuels when tested in the Ames Salmonella typhimurium assay, mouse lymphoma, and the rat bone marrow chromosomal aberration assay (Lebowitz et al. 1979). Unleaded gasoline did not induce unscheduled DNA synthesis in the male rat kidney at doses known to be nephrotoxic.

### **Carcinogenicity**

As indicated earlier, chronic gasoline exposures produces renal tumors in rats.

## **REGULATIONS AND STANDARDS**

The American Conference of Governmental Industrial Hygienists (ACGIH 1990) adopted a threshold limit value (TLV) of 300 ppm (mg/m<sup>3</sup>) for gasoline vapors. Because of the complexity



and variability in composition, OSHA has no standard but regulates the toxic components by their respective PELs (i.e., n-hexane, benzene, alkyl lead).

Gasoline as such is not mentioned in HEAST (1990) as having a specific cancer slope factor (CSF) or reference dose (RfD). However, individual components such as benzene, other aromatics, and n-hexane that have CSF or RfD values should be evaluated individually.

COMPOUND CAS NO.	ACGIH TLV ppm	RfD (inhal) mg/kg/day	RfD (oral) mg/kg/day	SLOPE FACTOR (inhal) mg/kg/day	SLOPE FACTOR (oral) mg/kg/day
Benzene 71-43-2	0.1	N/A	N/A	2.9E-2	2.9E-2
Ethylene dibromide 106-93-4	A2 <sup>1</sup>	N/A	N/A	7.6E-1	8.5E+1
Ethylene dichloride 107-06-2	10	N/A	N/A	9.1E-2	9.1E-2
n-Hexane 110-54-3	50	6E-1	2E-1	N/A	N/A
Tetraethyl lead 78-00-2	0.1 <sup>2</sup>	1E-7	2.9E-8	N/A	N/A

- <sup>1</sup> A2 - Substance classed as a suspected human carcinogen, no ACGIH TLV listed.  
<sup>2</sup> mg/m<sup>3</sup>, not ppm.

## REFERENCES

- ACGIH. 1990. Threshold Limit Values for Chemical Substances and Biological Exposure Indices for 1990-1991. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists.
- Donaldson, S.G. 1990. Volatilization of Gasoline from Contaminated Soil. M.S. Thesis, University of Reno.
- Hanis, N.M., T.M. Holmes, L.G. Shallenberger, and K.E. Jones. 1982. Epidemiologic Study of Refinery and Chemical Plant Workers. J. Occup. Med. 24:203-212.
- HEAST. 1990. Health Effects Assessment Summary Tables, Environmental Protection Agency. Office of Emergency and Remedial Response. EPA, OERR 9200 6-303, (90-4).

- Kaplan, S. 1986. Update of a Mortality Study of Workers in Petroleum Refineries. *J. Occup. Med.* 28:514-516.
- Korte, F. and W. Klein. 1982. Degradation of Benzene in the Environment. *Ecotox. Environ. Safety* 6:311-327.
- Lee, T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. *Lancet* 8134:149.
- Lebowitz, H., D. Brusick, and D. Matheson et al. 1979. Commonly Used Fuels and Solvents Evaluated in a Battery of Short-term Bioassays. *Environ. Mutagen.* 1:172-173.
- MacFarland, H.N. 1982. Chronic Gasoline Toxicology. In: *Proceedings of the Symposium: The Toxicology of Petroleum Hydrocarbons*, American Petroleum Institute, Washington, DC.
- McDermott, H. and S. Killiany. 1978. Quest for a Gasoline TLV. *Am. Ind. Hyg. Assoc. J.* 39:110-117.
- Song, H.G. 1988. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey.
- Thomas, T.L., R.J. Waxweiler, and R. Moure-Eraso et al. 1982. Mortality Patterns Among Workers in Three Texas Oil Refineries. *J. Occup. Med.* 24:135-141.
- Wong, O. 1987. An Industry Wide Mortality Study of Chemical Workers Occupationally Exposed to Benzene. *Br. J. Ind. Med.* 44:365-381.
- Von Burg, R. 1989. Toxicology Update for Gasoline. *J. Appl. Toxicol.* 9:203-210.

## **TOXICOLOGY PROFILE FOR WASTE OIL (RRPH)**

### **GENERAL DATA**

"Waste Oil" is a generic term commonly used to describe a range of petroleum compounds from heavy fuels to grease.

As much as one to two percent of the world's crude oil is refined to produce lubricating oil (24 million tons) (Vazquez-Duhalt 1989). The composition of waste oil is extremely variable and depends upon the original crude oil source, type of processing and refining, blending, additives, and use history. Waste oil may therefore range from virgin product accidentally spilled to used machine or automotive oil.

Petroleum oils are produced from the middle to heavy distillate fractions of crude oil. Because of the high boiling points for these fractions, the aromatic hydrocarbons benzene, ethylbenzene, toluene, and xylenes, typically found in lighter fuels will not be present in oils in high concentrations. These fractions may be further processed or treated to remove unwanted materials such as nitrogen, sulfur, metals, or polynuclear aromatic hydrocarbons (PAHs). For the most part, oils destined for the consumer market have been laundered to a very low content of PAHs. However, oils in refinery spills may contain several hundred ppm of PAHs. Used motor oil contains Pb, Zn, Cu, Cd, Cr, Ni, and other metals. Lead is the most abundant metal in motor oil and may be present at a concentration high as one percent (Vazquez-Duhalt 1989).

### **FATE AND TRANSPORT**

Oil with characteristics (e.g. vapor pressure, viscosity) closer to fuel oils may volatilize to some extent to the air. However, microbial degradation will, more than likely, be the primary mechanism for the mineralization of spilt material. As much as 90 percent of the material resembling jet fuel may be removed by a combination of evaporation and microbial degradation with a half-life of one to eight weeks; in contrast, heavier fractions that resemble bunker oil (C<sub>15</sub> and above) may be degraded only 25 to 30 percent and may be extremely persistent in soils (Song 1988). Given the correct circumstances, waste oils can percolate through the soil and float on the ground water. When spilled onto surface water, waste oils can be toxic to fish, waterfowl, and algae, but this is highly dependent upon the characteristics of the oily material and the size of the spill.

Approximately 30 percent of waste motor oil and lubricants produced are released into the environment. Because of the large quantities involved, the persistence of oil residues in the environment, and the potential for ecotoxicity, waste oils are an important environmental concern (Vazquez-Duhalt 1989).

When oil is spilt onto soil, it fills the spaces between the soil particles and hampers oxygen access, thereby promoting anaerobic zones. On the periphery of these oil-soiled zones, aerobic bacteria are promoted. Hence, these outer zones show increased nitrifying, denitrifying, ammonifying, and hydrocarbon-oxidizing microorganisms. The activity of these organisms in the outer zones increases the concentration of easily accessible substrates which stimulates an increase in the numbers of anaerobic nitrogen fixing bacteria (Vazquez-Duhalt 1989). Thus, under certain circumstances, oil addition to soil can function as an amendment and increase the productivity of the soil.

## **TOXICITY DATA**

### **Human Toxicology**

In general, most oily materials derived from petroleum have a low order of toxicity. Inhalation of components of waste oil at concentrations sufficient to achieve a level of intoxication is not likely at normal temperatures and pressures. An attempt to generate a kerosene (diesel) laden atmosphere only resulted in an ambient concentration of 14 ppm (Carpenter et al. 1976). However, under certain occupational settings like tank cleaning, it may be possible to generate mists or aerosols that can lead to symptoms of overexposure. These symptoms may include headache, dizziness, nausea, gastrointestinal symptoms, shortness of breath, weakness, confusion, drowsiness, and possibly death (HSDB 1991). A single report considered chronic repeated exposure to an oil mist for 17 years to be the cause of lipid pneumonia in workers "heavily" exposed (Proctor et al. 1989).

Ingestion of petroleum waste oil (other than fuel oil) either accidentally, intentionally, or from contaminated well-water is not expected to have a significant effect except possibly induction of gastrointestinal hypermotility and diarrhea (MacFarland et al. 1982), and nausea and vomiting. Ingestion of the heavier waste oils is not expected to be complicated by aspiration into the lung, which produces a potentially lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

### **Mammalian Toxicology and Significant Studies**

The acute oral and dermal LD<sub>50</sub> of petroleum waste oil is expected to be greater than 5 g/kg, or practically non toxic. Diesel fuel has an acute oral LD50 is in the range of 9 ml/kg body weight. New or used motor oil has an LD50 of 25 ml/kg, as does heavy fuel #6. Other properties, such as eye irritation, have ratings of practically non-irritating to mildly irritating. Skin irritation scores are similarly low ranging from non-irritating to mildly irritating (Beck et al. 1982). There are no readily available reports on hypersensitivity responses to waste oils, but sensitization is an expected effect because refined products in this distillation range have been reported to produce hypersensitivity reactions (Beck et al. 1982). Dermal absorption of oil can also be expected but the oil itself is unlikely to be the cause of systemic toxicity. Any toxicity is more likely to be attributable to a concomitant absorption of some oil contaminant.

Exposure of CD-1 mice to diesel vapor for eight hours per day on five consecutive days resulted in a decrement of performance on the roto-rod test, square box activity test, and hot plate test. However, the corneal reflex and inclined plane test was unaffected. General observations included vasodilation, ataxia, poor grooming, and in some cases tremor (Kainz and White 1982).

Exposure of rats to aerosolized diesel fuel at concentrations up to 6 mg/L produced direct toxic effects on the lungs but did not produce any neurotoxicity (Dalbey et al. 1987).

### **Reproductive Toxicity**

Female rats were exposed 6 hours per day to air concentrations of 0, 100, and 400 ppm during days 6 through 15 of gestation. Neither jet fuel or No. 2 Fuel Oil produced any significant detrimental effects on the reproductive parameters of the experimental animals (Beliles and Mecler 1982). Neither Jet Fuel A or diesel fuel at exposure levels of 400 ppm, six hours per day, five days per week, for eight weeks reduced the fertility of CD-1 male mice (API 1980a,b).

External application of new or used motor oil to the egg shell of a number of bird species caused embryotoxicity and lethality. The used motor oil was more toxic than the new motor oil (Hoffman et al. 1982).

### **Genotoxicity**

Ames testing of several common fuel oils produced mainly negative results. However, the "Modified Ames Assay", introduced by Blackburn et al. (1988), did demonstrate mutagenicity in two straight run gas oils that were previously considered to be negative. Diesel fuel was negative in a mouse lymphoma assay but positive on the rat bone marrow cytogenetics assay when administered by intraperitoneal injection (Conaway et al. 1982). Heating oil #2 did produce a positive Ames test and positive results in two other short term bioassays (Rothman and Emmett 1988).

Used motor oil has been shown to be highly mutagenic to Salmonella bacteria (Peake and Parker 1980). New crankcase motor oil initially tested negative with the standard Ames Assay but after an extraction procedure to remove "interfering chemicals", a dose dependent mutagenic response was observed with both gasoline and diesel crankcase oils. The extracts of the new motor oils, however, are considerably less mutagenic than the Used Crankcase Oil extracts. This effect can be explained by the fact that, during engine operation, the oil accumulates combustion dust and PAH formed in the combustion process or directly from the fuel (Thony et al. 1975). Extracts from the diesel and gasoline type engines were about equally potent (Dutcher et al. 1986).

### **Carcinogenicity**

In classical mouse skin-painting bioassays, all petroleum fractions derived from a crude oil source that boiled between 120°F and 700°F showed a low level of tumorigenic activity (Lewis et al. 1982). Home heating oil also showed a low degree of tumorigenicity in a more recent mouse skin-painting assay (Witschi et al. 1987). Topical application of used motor oil from gasoline driven vehicles increased the incidence of local tumors in a dose related fashion. The application of new motor oil to mouse skin did not induce skin tumors (Saffiotti and Shubik 1963). This information plus the demonstrated mutagenic potential of used motor oils and their PAH content, allows a determination that such oils can be considered to be potentially carcinogenic (IARC 1984).

In a case referent study, Seimietycki et al. (1987) reported an increase of several specific cancers associated with exposures to different petroleum products. Leaded gasoline was associated with stomach cancer; aviation gasoline with kidney cancer; diesel fuel with non adenocarcinoma of the lung and prostate cancer; and mineral spirits with squamous cell lung cancer. However, not all parameters of concern were properly controlled, excluded, or assessed, making conclusions from this study unreliable.

IARC (1989) has classified gasoline, diesel fuel, and residual oil Category 2B, having limited evidence of carcinogenicity in animals and inadequate evidence in humans. Used motor oil (crankcase oil) is also classified as a category 2B. Light fuel oils, crude oil, and jet fuels have been classified as Category 3, having inadequate evidence of carcinogenicity in either animals or humans.

## REGULATIONS AND STANDARDS

Neither the American Conference of Governmental Industrial Hygienists (ACGIH) nor OSHA have recommended or established permissible exposure standards (PELs) for diesel fuels or waste oils. NIOSH has recommended a 10 hour TWA of 100 mg/m<sup>3</sup> for kerosene or 14 ppm (MMWR. 37:24). The ACGIH (1991) and OSHA (1985) recommend a TLV of 5 mg/m<sup>3</sup> for Oil Mists.

Diesel fuels are not mentioned in HEAST (1990), nor identified for a specific cancer Slope Factor (CSF) or reference dose (RfD). However, individual components such as benzene, other aromatics, and n-hexane that have CSF or RfD values should be evaluated individually.

## REFERENCES

- API. 1980a. Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-03. American Petroleum Institute Medical Research Publications, Washington, D.C.
- API. 1980b. Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-04. American Petroleum Institute Medical Research Publications, Washington, D.C.
- Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Beliles, R.P. and F.J. Mecler. 1982. Inhalation Teratology of Jet Fuel, Fuel Oil and Petroleum Naphtha in Rats. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Blackburn, G.R., R.A. Deitch, and T.A. Roy et al. 1988. Estimation of the Dermal Carcinogenic Potency of Petroleum Fractions Using a Modified Ames Assay. In: Polynuclear Aromatic Hydrocarbons: A Decade of Progress, Proceedings of the Tenth International Symposium. M. Cooke and A.J. Dennis, editors. Battelle Press, Columbus, Ohio. pp. 83-97.
- Carpenter, C.P., D.L. Geary, and R.C. Meyers et al. 1976. Petroleum Hydrocarbon Toxicity Studies XI. Animal and Human Response to Vapors of Deodorized Kerosene. Toxicol. Appl. Pharmacol. 36:443-456.
- Clayton, G.D. and F.E. Clayton. 1982. Patty's Industrial Hygiene and Toxicology. 3rd Edition. G.D. Clayton and F.E. Clayton, editors. John Wiley and Sons, Publishers, New York, New York.
- Conaway, C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity Evaluation of Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

- Dalbey, W., M. Henry, and R. Holmberg et al. 1987. Role of Exposure Parameters in the Toxicity of Aerosolized Diesel Fuel in the Rat. *J. Appl. Toxicol.* 7:265-275.
- Dutcher, S.J., A.P. Li, and R.O. McClellan. 1986. Mutagenicity of Used Crankcase Oils from Diesel and Spark Ignition Automobiles. *Environ. Res.* 40:155-163.
- HEAST. 1990. Health Effects Assessment Summary Tables, EPA, OERR 9200 6-303, (90-4).
- Hoffman, D.J., W.C. Eastin, and M.L. Gay. 1982. Embryotoxic and Biochemical Effects of Waste Crankcase Oil on Bird Eggs. *Toxicol. Appl Pharmacol.* 63:230-241.
- HSDB. 1990. Hazardous Substances Data Bank, National Library of Medicine. Washington, D.C.
- IARC. 1984. International Agency for Research on Cancer. Vol 33. Polynuclear Aromatic Hydrocarbons: Part 2. Carbon Black, Mineral Oils and Some Nitroarenes. World Health Organization, Lyon, France.
- IARC. 1989. International Agency for Research on Cancer. Vol 45. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. World Health Organization, Lyon, France.
- Kainz, R.J. and L.E. White. 1982. Consequences Associated with the Inhalation of Uncombusted Diesel Vapor. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- Kirk Othmer. 1984. Diesel Fuel. In: *Kirk-Othmer Encyclopedia of Chemical Technology*. F. Mark, M. Grayson, and D. Eckroth et al., Editors. 3rd Edition. John Wiley and Sons, New York.
- Kryzanovskij, N.V. 1971. Occupational Health Conditions Associated with the Cleaning of Oil Tankers and their Effect on Worker's Health. *Gig. Tr. Prof. Zabol.* 15:14-17.
- Lee T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. *The Lancet* 8134:149.
- Lebowitz H., D. Brusick, and D. Matheson et al. 1979. Commonly Used Fuels and Solvents Evaluated in a Battery of Short-term Bioassays. *Environ. Mutagen.* 1:172-173.
- Lewis, S.C., R.W. King, S.T. Cragg, and D.W. Hillman. 1982. Skin Carcinogenic Potential of Petroleum Hydrocarbons. 2. Carcinogenesis of Crude Oil, Distillate Fractions and Chemical Class Subfractions. In: *Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons*. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.
- NIOSH. 1977. Criteria for a Recommended Standard...Occupational Exposure to Refined Petroleum Solvents. National Institute for Occupational Safety and Health, Cincinnati, Ohio. NIOSH Publication #77-192.

OHM TADS. 1991. Oil and Hazardous Materials - Technical Assistance Data Systems. Environmental Protection Agency, Emergency Response Division, Washington D.C.

Peake, E. and K. Parker. 1980. Polynuclear Aromatic Hydrocarbons and the Mutagenicity of Used Crankcase Oil. In: A. Bjorseth and J. Dennis, editors. Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects. Battelle Press, Columbus, Ohio. pp 1025-1039.

Rothman, N. and E.A. Emmett. 1988. The Carcinogenic Potential of Spetroleum-derived Products. Chapter 7. In: Occupational Medicine: State of the Art Reviews. 3:475-482.

Saffiotti, U. and P. Shubik. 1963. Studies on Promoting Action in Skin Carcinogenesis. National Cancer Inst. Monogr. 10:489-507.

Siemiatycki, J., R. Dewar, and L. Nadon et al. 1987. Associations Between Several Sites of Cancer and Twelve Petroleum Derived Liquids. Scan. J. Work Environ. Health. 13:493-504.

Song, H.G. 1988. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community.

Thony, C., J. Thony, and M. Lafontaine et al. 1975. Concentration En Hydrocarbons Polycycliques Aromatiques Concerogenes Des Huiles Minerale. Arch. Mal. Prof. Med. Travail Sec. Soc. 36: 281-300.

Vazquez-Duhalt, R. 1989. Environmental Impact of Used Motor Oil. Sci. Total Environ. 79:1-23.

Witschi, H.P., L.H. Smith, and E.L. Frome et al. 1987. Skin Tumorigenic Potential of Crude and Refined Coal Liquids and Analogous Petroleum Products. Fund. Appl. Toxicol. 9:297-303.



## TETRACHLOROETHENE

Tetrachloroethene is absorbed following inhalation (IARC 1979) and oral (EPA 1985a,b) exposure. Tetrachloroethene vapors and liquid also can be absorbed through the skin (EPA 1985a,b). The principal toxic effects of tetrachloroethene in humans and animals following acute and longer-term exposures include CNS depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage (EPA 1985a,b; Buben and O'Flaherty 1985). Humans exposed to doses of between 136 and 1,018 mg/m<sup>3</sup> for 5 weeks develop central nervous system effects, such as lassitude and signs of inebriation (Stewart et al. 1974). The offspring of female rats and mice exposed to high concentrations of tetrachloroethene for 7 hours daily on days 6-15 of gestation developed toxic effects, including a decrease in fetal body weight in mice, and a small but significant increase in fetal resorption in rats (Schwetz et al. 1975). Mice also exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al. 1975). In a National Cancer Institute bioassay (NCI 1977), an increased incidence of hepatocellular carcinoma was observed in both sexes of B6C3F1 mice administered tetrachloroethylene in corn oil by gavage for 78 weeks. Increased incidence of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have been observed in long term bioassays in which rats were exposed to tetrachloroethene by inhalation (NTP 1986).

Tetrachloroethene is currently under review by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) and estimates of cancer potency were recently withdrawn by EPA (1992b). However, the EPA Environmental Criteria and Assessment Office (ECAO) (1992a) currently classifies tetrachloroethene as a Group B2/C carcinogen (Probable/Possible Human Carcinogen). ECAO (1992a) has reported an oral slope factor of  $5.2 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$  based on liver tumors observed in the NCI (1977) gavage bioassay for mice. An inhalation cancer unit risk of  $5.8 \times 10^{-7} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$  is based on an NTP (1986) bioassay in rats and mice in which leukemia and liver tumors were observed (ECAO 1992a). Both the cancer slope factor and unit risk are currently under review by EPA. EPA (1993) also derived an oral RfD of  $1 \times 10^{-2} \text{ mg/kg/day}$  for tetrachloroethene based on a six-week gavage study by Buben and O'Flaherty (1985). In this study, liver weight/body weight ratios were significantly increased in mice and rats treated with 71 mg/kg-day tetrachloroethene but not in animals treated with 14 mg/kg-day. The RfD was derived using a NOAEL of 14 mg/kg/day and applying an uncertainty factor of 1,000. EPA (1992b) established a subchronic oral RfD of  $1 \times 10^{-1} \text{ mg/kg/day}$ , using an uncertainty factor of 100 based on the same study and effect of concern.

The American Conference of Governmental Industrial Hygienists (ACGIH) has set a Short-Term Exposure Level - Threshold Limit Value of 200 ppm (1,000 mg/m<sup>3</sup>) for tetrachloroethene (ACGIH 1991). The STEL-TLV is defined as a 15-minute time-weighted average which should not be exceeded at any time during a work day. A health criterion for acute inhalation exposure to tetrachloroethene of 100 mg/m<sup>3</sup> can be derived from the STEL-TLV by combining it with a safety factor of 10 to account for the healthy worker effect which assumes that employed persons are healthier than the general population.

## REFERENCES

American Conference of Governmental Industrial Hygienist (ACGIH). 1991. Threshold Limit Values and Biological Exposure Indices for 1990-1991. Cincinnati, Ohio.

- Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the Role of Metabolism in the Hepatotoxicity of Trichloroethylene and Perchloroethylene: A Dose-effect Study. *Toxicol. Appl. Pharmacol.* 78:105-122.
- Environmental Protection Agency (EPA). 1985a. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Health and Environmental Assessment, Washington, D.C. July 1985. EPA 600/8-82-005F.
- Environmental Protection Agency (EPA). 1985b. Drinking Water Criteria Document for Tetrachloroethylene. Office of Drinking Water, Criteria and Standards Division, Washington, D.C. June 1985.
- Environmental Criteria and Assessment Office (ECAO). 1992a. Written Correspondence from Joan Dollarhide, Chemical Mixtures Assessment Branch, U.S. Environmental Protection Agency. July 16, 1992.
- Environmental Protection Agency (EPA). 1992b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Remedial Response, Washington, D.C. FY-1992.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- International Agency for Research on Cancer (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France.
- National Cancer Institute (NCI). 1977. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13, Washington, D.C. DHEW (NIH) Publication No. 77-813.
- National Toxicology Program (NTP). 1986. Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP Technical Report Series No. 311, Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567.
- Schwetz, B.A., B.K.J. Leong, and P.J. Gehring. 1975. The Effect of Maternally Inhaled Trichloroethylene, Perchloroethylene, Methyl Chloroform, and Methylene Chloride on Embryonal and Fetal Development in Mice and Rats. *Toxicol. Appl. Pharmacol.* 55:207-219.
- Stewart, R.D., C.L. Hake, H.V. Forster, A.J. Lebrun, J.F. Peterson, and A. Wu. 1974. Tetrachloroethylene: Development of a Biologic Standard for the Industrial Worker by Breath Analysis. Medical College of Wisconsin, Milwaukee, Wisconsin. NIOSH-MCOW-ENUM-PCE-74-6.

## BARIUM

Barium and compounds are absorbed to a limited extent following oral and inhalation exposures (ATSDR 1992). Respiratory weakness, paralysis, hypertension, and abnormalities in heart rhythm have been frequently observed following acute ingestion (ATSDR 1992). In occupationally exposed workers, inhalation of barium sulfate has been associated with a minor lung effect called baritosis, a benign pneumoconiosis (Doig 1976; Goyer 1986). The effects of elevated barium levels in drinking water on blood pressure, hypertension, stroke, heart disease, and altered electrocardiograms have been investigated in several epidemiological studies. No adverse effects were found with barium concentrations as high as 10 mg/L (0.21 mg/kg/day) (Brenniman and Levy 1984, 1985; Brenniman et al. 1979a,b, 1981; Wones et al. 1990). Subchronic and chronic oral exposure of rats to barium (as high as 7.1 mg/kg/day for one month and as low as 0.54 mg/kg/day for 16 months), however, resulted in increased blood pressure and cardiovascular cellular changes (Kopp et al. 1985; Perry et al. 1983, 1985, 1989). One animal study did not result in adverse effects at the highest dose tested of 15 mg/kg/day (McCauley et al. 1985); two other studies did not indicate adverse effects but did not measure blood pressure (Schroeder and Mitchener 1975a,b; Tardiff et al. 1980). Subchronic inhalation of barium carbonate dust (3.6 mg/m<sup>3</sup>) by experimental animals has been associated with reduced sperm count, increased fetal mortality, atresia of the ovarian follicles, decreased body weight, and alterations in liver function (Tarasenko et al. 1977).

EPA (1993a) derived an oral reference dose (RfD) based on a weight-of-evidence approach that takes into account recent findings of two human epidemiologic studies (Brenniman and Levy 1984; Wones et al. 1990) as well as various rodent studies (Perry et al. 1983; McCauley et al. 1985; Schroeder and Mitchener 1975a,b; Tardiff et al. 1980). No single study was appropriate to calculate a lifetime RfD for barium (EPA 1993a). A LOAEL was not identified in either of the two epidemiological studies; however, the effect of concern in the animal studies was high blood pressure. Using a NOAEL of 0.21 mg/kg/day (Wones et al. 1990) and an uncertainty factor of three, an oral RfD of  $7 \times 10^{-2}$  mg/kg/day was calculated. The chronic RfD was adopted as a subchronic RfD by EPA (1993b). EPA (1993b) has also developed chronic and subchronic inhalation RfCs of  $5 \times 10^{-4}$  and  $5 \times 10^{-3}$  mg/m<sup>3</sup> for barium based on a study by Tarasenko et al. (1977). In this study, rats were exposed to barium carbonate dust at airborne concentrations of up to 5.2 mg/m<sup>3</sup> for 4-6 months. Adverse effects noted at this concentration included decreased body weight, alterations in liver function, and increased fetal mortality. Uncertainty factors of 1,000 and 100 were used in developing the chronic and subchronic RfCs, respectively.

## REFERENCES

- Agency for Toxic Substances and Disease Control (ATSDR). 1992. Toxicological Profile for Barium and Compounds. U.S. Public Health Center, Center for Disease Control, Research Triangle Park, North Carolina.
- Brenniman G.R. and P.S. Levy. 1984. High Barium Levels in Public Drinking Water and its Association with Elevated Blood Pressure. In: *Advances in Modern Toxicology IX*, E.J. Calabrese, Ed. Princeton Scientific Publication, Princeton, New Jersey. p 231-249.
- Brenniman G.R. and P.S. Levy. 1985. Epidemiological Study of Barium in Illinois Drinking Water Supplies. In: E.J. Calabrese, R.W. Tuthill, and L. Condie, eds. *Inorganics in Water and Cardiovascular Disease*. Princeton, New Jersey: Princeton Scientific Publishing Co. p. 231-240.

- Brenniman, G.R., W.H. Kojola, and P.S. Levy et al. 1979a. Health Effects of Human Exposure to Barium in Drinking Water. Cincinnati, Ohio: U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory. EPA/600/1-79/003.
- Brenniman, G.R., T. Namekata, and W.H. Kojola et al. 1979b. Cardiovascular Disease Death Rates in Communities with Elevated Levels of Barium in Drinking Water. Environ. Res. 20:318-324.
- Brenniman, G.R., W.H. Kojola, and P.S. Levy et al. 1981. High Barium Levels in Public Drinking Water and its Association with Elevated Blood Pressure. Arch. Environ. Health 36:28-32.
- Doig, A.T. 1976. Baritosis: A Benign Pneumoconiosis. Thorax 31:30-39.
- Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-021.
- Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.
- Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.
- Goyer, R.A. 1986. Toxic Effects of Metals. In C.D. Klaassen, M.D. Amdur, and J. Doull, eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 3rd Ed. Macmillan Publishing Co., New York. Pp. 623-624.
- Kopp, S.J., H.M. Perry, Jr., and J.M. Feliksik et al. 1985. Cardiovascular Dysfunction and Hypersensitivity to Sodium Pentobarbital Induced by Chronic Barium Chloride Ingestion. Toxicol. Appl. Pharmacol. 77:303-314.
- Mc Cauley, P.T., B.H. Douglas, R.D. Laurie, and R.J. Bull. 1985. Investigations into the Effect of Drinking Water Barium on Rats. Environ. Health. Perspect. Vol. IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, New Jersey. p. 197-210.
- Perry, H.M., S.J. Kopp, M.W. Erlanger, and E.G. Perry. 1983. XVII. Cardiovascular Effects of Chronic Barium Ingestion. In D.D. Hemphill, ed. Proceedings of the University of Missouri's 17th Annual Conference of Trace Substances in Environmental Health. University of Missouri Press, Columbia, Missouri (As cited in EPA 1984).
- Perry, H.M. Jr., E.F. Perry, and M.W. Erlanger et al. 1985. Barium-Induced Hypertension. Adv. Mod. Environ. Toxicol. 9:221-229.
- Perry, H.M. Jr., S.J. Kopp, and E.F. Perry et al. 1989. Hypertension and Associated Cardiovascular Abnormalities Induced by Chronic Barium Feeding. J. Toxicol. Environ. Health 28:373-388.

- Schroeder, H.A. and M. Mitchener. 1975a. Life-Term Effects of Mercury, Methyl Mercury and Nine Other Trace Metals on Mice. *J. Nutr.* 105:452-458.
- Schroeder, H.A. and M. Mitchener. 1975b. Life-Term Studies in Rats: Effects of Aluminum, Barium, Beryllium and Tungsten. *J. Nutr.* 105:421-427.
- Tarasenko, M., O. Promin, and A. Silayev. 1977. Barium Compounds as Industrial Poisons (An Experimental Study). *J. Hyg. Epiderm. Microbial. Immunol.* 21:361 (As cited in EPA 1984).
- Tardiff, R.G., M. Robinson, and N.S. Ulmer. 1980. Subchronic Oral Toxicity of BaCl<sub>2</sub> in Rats. *J. Environ. Pathol. Toxicol.* 4:267-275.
- Wones, R.G., B.L. Stadler, and L.A. Frohman. 1990. Lack of Effect of Drinking Water Barium on Cardiovascular Risk Factor. *Environ Health Perspective.* 85:1-13.

## MANGANESE

Manganese is considered to be an essential element and among the least toxic of the trace metals (NRC 1989). The oral absorption of dietary manganese ranges from 3 to 10 percent (EPA 1993). However, manganese is absorbed to a greater extent following inhalation exposures. The National Research Council has established a provisional recommended dietary allowance for adults of 2 to 5 mg/day (NRC 1989). The effects following acute exposure to manganese are unknown.

Chronic occupational exposure to manganese dust ( $0.02 - 2.6 \text{ mg/m}^3$ ) has been associated with respiratory symptoms and pneumonitis (Chandra et al. 1981, 1990). Higher levels have been associated with a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking. For example, male workers exposed to manganese dioxide, tetroxide, and various salts [time-weighted-average (TWA) of total airborne manganese dust ranged from  $0.07 - 8.61 \text{ mg/m}^3$ ] experienced an increased incidence of psychomotor disturbances (e.g., reaction time, hand-eye coordination, and hand steadiness) (Roels et al. 1987). Other effects observed in humans occupationally exposed to manganese dust include hematological (Chandra et al. 1981; Flinn et al. 1941; Kesic and Hausler 1954), cardiovascular (Saric and Hrustic 1975), and reproductive effects (Cook et al. 1974; Emara et al 1971; Lauwerys et al 1985; Rodier 1955).

In adults, a safe intake of manganese from dietary sources ranges from 2-10 mg/day (10 mg/day =  $0.14 \text{ mg/kg/day}$ ) (WHO 1973; NRC 1989; Schroeder et al. 1966). Individuals who chronically ingested drinking water from natural wells containing manganese concentrations of 1,600 to 2,300  $\mu\text{g/L}$  ( $0.06 \text{ mg/kg/day}$ ), showed a statistically significant increase in minor neurologic effects (neurologic exam scores) (Kondakis et al. 1989). Higher concentrations in drinking water ( $0.8 \text{ mg/kg/day}$ ) have resulted in symptoms including lethargy, increased muscle tonus, tremor, and mental disturbances (Kawamura et al. 1941).

The apparent differences in manganese toxicity following dietary and drinking water exposures can be attributed to the greater bioavailability of manganese from water (EPA 1993). Chronic oral exposure of rats to manganese chloride can also result in central nervous system dysfunction (Leung et al. 1981; Lai et al. 1982). Chronic inhalation exposure of experimental animals (monkeys, rats, mice, hamsters) has resulted in respiratory effects, however, other studies have demonstrated that these effects may be immunological in origin (ATSDR 1992).

Manganese has not been reported to be teratogenic but it has been observed to cause depressed reproductive performance and reduced fertility in humans and experimental animals (EPA 1984a). Certain manganese compounds have been shown to be mutagenic in a variety of bacterial tests. Manganese chloride and potassium permanganate can cause chromosomal aberrations in mouse mammary carcinomal cells. Manganese was moderately effective in enhancing viral transformation of Syrian hamster embryo cells (EPA 1984a,b).

EPA (1993a) established a weight-of-evidence classification for manganese of D (not classifiable as to human carcinogenicity). EPA (1993a) derived two separate oral reference doses (RfD). The separate RfDs for food and water indicate a potentially higher bioavailability of manganese from drinking water than from the diet. The RfD associated with oral exposure to drinking water is  $5 \times 10^{-3} \text{ mg/kg/day}$  based on a no observed adverse effect level (NOAEL) of  $5 \times 10^{-3} \text{ mg/kg/day}$  for humans (Kondakis et al. 1989). EPA (1993a) also derived an RfD of  $1.4 \times 10^{-1} \text{ mg/kg/day}$  for manganese in food based on a NOAEL of  $0.14 \text{ mg/kg/day}$  (10 mg/day) in humans chronically

exposed to dietary levels (WHO 1973; Schroeder et al. 1966; NRC 1989). The effect of concern was on the central nervous system, and an uncertainty factor of one was used to derive both RfDs. The chronic RfD in food was adopted as the subchronic RfD (EPA 1993b). EPA (1993a) derived a chronic inhalation reference concentration (RfC) of  $4 \times 10^{-4}$  mg/m<sup>3</sup> based upon an occupational study conducted by Roels et al. (1987) in which respiratory symptoms and psychomotor disturbances were observed. EPA (1993b) adopted the chronic RfC as the subchronic RfC. An uncertainty factor of 900 was used to derive both RfCs.

## REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for Manganese. U.S. Department of Health and Human Services, Center for Disease Control.
- Chandra, S.V., G.S. Shukla, R.S. Striavastava, H. Singh, and V.P. Gupta. 1981. An Exploratory Study of Manganese Exposure to Welders. Clin. Toxicol. 18:407-416.
- Cook, D.G., S. Fahn, and K.A. Brait. 1974. Chronic Manganese Intoxication. Arch. Neurol. 30:59-64.
- Emara, A.M., S.H. El-Ghawabi, O.I. Madkour, and G.H. El-Sarma. 1971. Chronic Manganese Poisoning in the Dry Battery Industry. Br. J. Ind. Med. 28:78-82.
- Environmental Protection Agency (EPA). 1984a. Health Assessment Document for Manganese. Final Report. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, Ohio. August 1984. EPA 600/8-83-013F.
- Environmental Protection Agency (EPA). 1984b. Health Effects Assessment for Manganese (and compounds). Environmental Criteria and Assessment Office, Washington, D.C. EPA 540/1-86-057.
- Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.
- Flinn, R.H., P.A. Neal, and W.B. Fulton. 1941. Industrial Manganese Poisoning. J. Ind. Hyg. Toxicol. 23:374-387.
- Iregren, A. 1990. Psychological Test Performance in Foundry Workers Exposed to Low Levels of Manganese. Neurotox. Teratol. 12:673-675.
- Kawamura, R., H. Ikuta, and S. Fukuzumi et al. 1941. Intoxication by Manganese in Well Water. Kitasato Arch. Exp. Med. 18:145-149.
- Kesic, B. and V. Hausler. 1954. Hematological Investigation on Workers Exposed to Manganese Dust. Arch. Ind. Hyg. Occup. Med. 10:336-343.

- Kondakis, X.G., M. Makris, and M. Leotsinidis et al. 1989. Possible Health Effects of High Manganese Concentration in Drinking Water. *Arch. Environ. Health* 44:175-178.
- Lai, J.C.K., T.K.C. Leung, and L. Lim. 1982. Activities of the Mitochondrial NAD-Linked Isocitric Dehydrogenase in Different Regions of the Rat Brain. Changes in Aging and the Effect of Chronic Manganese Chloride Administration. *Gerontology* 28:81-85.
- Lauwerys, R., H. Roels, and P. Genet et al. 1985. Fertility of Wale Workers Exposed to Mercury Vapor or to Manganese Dust: A Questionnaire Study. *Am. J. Ind. Med.* 7:171-176.
- Leung, T.K.C., J.C.K. Lai, and L. Lim. 1981. The Regional Distribution of Monoamine Oxidase Activities Towards Different Substrates: Effects in Rat Brain of Chronic Administration of Manganese Chloride and of Aging. *J. Neurochem.* 36:2037-2043.
- National Research Council (NRC). 1989. Recommended Dietary Allowances, 10th Ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, D.C. 230-235.
- Rodier, J. 1955. Manganese Poisoning in Moroccan Miners. *Br. J. Ind. Med.* 12:21-35.
- Roels, H., R. Lauwerys, and J.P. Buchet et al. 1987. Epidemiological Survey Among Workers Exposed to Manganese: Effects on Lung, Central Nervous System, and Some Biological Indices. *Am. J. Ind. Med.* 11:307-327.
- Saric, M. and O. Hrustic. 1975. Exposure to Airborne Manganese and Arterial Blood Pressure. *Environ. Res.* 10:314-318.
- Schroeder, H.A., D.D. Balassa, and I.H. Tipton. 1966. Essential Trace Metals in Man: Manganese, a Study in Homeostasis. *J. Chron. Dis.* 19:545-571.
- WHO (World Health Organization). 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. 34-36.



## BENZENE

Benzene is readily absorbed following oral and inhalation exposure (EPA 1985). The toxic effects of benzene in humans and other animals following exposure by inhalation include central nervous system effects, hematological effects, and immune system depression. In humans, acute exposures to high concentrations of benzene vapors have been associated with dizziness, nausea, vomiting, headache, drowsiness, narcosis, coma, and death (NAS 1976). Chronic exposure (at least 20 years of worker exposure) to benzene vapors (1-100 ppm 8-hour TWA) reduce leukocyte, platelet, and red blood cell counts (EPA 1993). Benzene induced tumors of the zymal gland, oral cavity, leukemia and lymphoma in rodents chronically exposed by gavage to doses in the range of 25-500 mg/kg/day (Huff et al. 1989; NTP 1986; Maltoni et al. 1989). Many studies have also described a causal relationship between exposure to benzene by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC 1982; Rinsky et al. 1981; Ott et al. 1978; Wong et al. 1983).

Applying EPA's criteria for evaluating the overall evidence of carcinogenicity to humans, benzene is classified in Group A (Human Carcinogen) based on adequate evidence of carcinogenicity from epidemiological studies. EPA (1993) derived an oral cancer slope factor of  $2.9 \times 10^{-2}$  (mg/kg/day)<sup>-1</sup> and an inhalation unit risk of  $8.3 \times 10^{-6}$  (ug/m<sup>3</sup>)<sup>-1</sup> for benzene. These values were based on several studies in which increased incidence of nonlymphocytic leukemia were observed in humans occupationally exposed to benzene principally by inhalation (Rinsky et al. 1981; Ott et al. 1978; Wong et al. 1983). Equal weight was given to cumulative dose and weighted cumulative dose as well as to relative and absolute risk model forms (EPA 1993). EPA (1993) is currently reviewing both oral and inhalation RfDs for benzene, for which the status is pending.

The National Research Council's Committee on Toxicology has set a one-hour Emergency Exposure Guidance Level (EEGL), for benzene at 50 ppm (200 mg/m<sup>3</sup>) (NRC 1986). Formerly known as EEL, the EEGL is defined as a ceiling limit for an unpredicted single exposure lasting one to 24 hours, which is expected to be rare in the lifetime of any person. It is designed to avoid substantial decrements in performance during emergencies and takes into account the statistical likelihood of a non-incapacitative, reversible effect in exposed populations (NRC 1986). A health criterion for acute inhalation exposure to benzene of 20 mg/m<sup>3</sup> can be derived from the EEGL by combining it with a safety factor of 10 to account for the healthy worker effect, which assumes employed persons are generally healthier than the general population.

## REFERENCES

- Environmental Protection Agency (EPA). 1985. Drinking Water Criteria Document for Benzene (Final Draft). Office of Drinking Water, Washington, D.C. April 1985.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Huff, J.E., J.K. Haseman, and D.M. Demarini et al. 1989. Multiple-Site Carcinogenicity of Benzene in Fischer 344 Rats and B6C3F1 Mice. Environ. Health Perspect. 82:125-163.

- International Agency for Research On Cancer (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. Vol. 27.
- Maltoni, C., B. Conti, and G. Cotti et al. 1989. Benzene: A Experimental Multipotential Carcinogen. Results of Long-Term Bioassays Performed at the Bologna Institute of Oncology. Environ. Health Perspect. 82:109-124.
- National Academy of Science (NAS). 1976. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington, D.C.
- National Research Council (NRC). 1986. Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants. Volume 6. Prepared by the Committee on Toxicology. National Academy Press, 1986.
- National Toxicology Program (NTP). 1986. NTP-Technical Report Series No. 289. Toxicology and Carcinogenesis Studies of Benzene in F344/N Rats and B6C3F1 Mice (Gavage Studies). Research Triangle Park, North Carolina: U.S. Department of Health and Human Services, Public Health Service, National Institute of Health. NIH Publication No. 86-2545.
- Ott, M.G., J.C. Townsend, W.A. Fishbeck, and R.A. Langner. 1978. Mortality Among Individuals Occupationally Exposed to Benzene. Arch. Environ. Health 33:3-10.
- Rinsky, R.A., R.J. Young, and A.B. Smith. 1981. Leukemia in Benzene Workers. Am. J. Ind. Med. 3:217-245.
- Wong, O., R.W. Morgan, and M.D. Whorton. 1983. Comments on the NIOSH Study of Leukemia in Benzene Workers. Technical Report Submitted to Gulf Canada, Ltd., by Environmental Health Associates.

## TRICHLOROETHENE

Absorption of trichloroethene (TCE) from the gastrointestinal tract is virtually complete. Absorption following inhalation exposure is proportional to concentration and duration of exposure (EPA 1985). TCE is a CNS depressant following acute and chronic exposures. In humans, single oral doses of 15 to 25 ml (21 to 35 grams) of TCE have resulted in vomiting and abdominal pain, followed by transient unconsciousness (Stephens 1945). High-level exposure can result in death due to respiratory and cardiac failure (EPA 1985). Hepatotoxicity has been reported in human and animal studies following acute exposure to TCE (EPA 1985). Nephrotoxicity has been observed in animals following acute exposure to TCE vapors (ACGIH 1986; Torkelson and Rowe 1981). Subacute inhalation exposures of mice have resulted in transient increased liver weights (Kjellstrand et al. 1983a,b). Industrial use of TCE is often associated with adverse dermatological effects including reddening and skin burns on contact with the liquid form, and dermatitis resulting from vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported following exposure to TCE in dilute, aqueous solutions (EPA 1985). TCE has caused significant increases in the incidence of hepatocellular carcinomas in mice (NCI 1976) and renal tubular-cell neoplasms in rats exposed by gavage (NTP 1983), and pulmonary adenocarcinomas in mice following inhalation exposure (Fukuda et al. 1983; Maltoni et al. 1986). TCE was mutagenic in *Salmonella typhimurium* and in *E. coli* (strain K-12), utilizing liver microsomes for activation (Greim et al. 1977).

EPA is currently reviewing the carcinogenicity of TCE. The EPA ECAO currently classifies TCE as a Group B2/C--Probable/Possible Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies. ECAO (1992) reported an oral cancer potency factor of  $1.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$  based on two gavage studies conducted in mice in which increased incidence of liver tumors were observed (Maltoni et al. 1986; Fukuda et al. 1983). An inhalation cancer unit risk of  $1.7 \times 10^{-6} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$  has been derived for TCE based on an increased incidence of lung tumors in mice exposed via inhalation (ECAO 1992; NCI 1976). The cancer estimates are currently under review by EPA. EPA (1987) developed an oral RfD of  $7.35 \times 10^{-3} \text{ mg/kg/day}$  based on a subchronic inhalation study in rats in which elevated liver weights were observed following exposure to 55 ppm, 5 days/week for 14 weeks (Kimmerle and Eben 1973). A safety factor of 1,000 was used to calculate the RfD. However, this RfD is currently under review by EPA.

The National Research Council's Committee on Toxicology has set a one-hour EEGL for trichloroethene at 200 ppm ( $1,000 \text{ mg/m}^3$ ) (NRC 1988). Formerly known as EEL, the EEGL is defined as a ceiling limit for an unpredicted single exposure lasting one to 24 hours, which is expected to be rare in the lifetime of any person. It is designed to avoid substantial decrements in performance during emergencies and takes into account the statistical likelihood of a non-incapacitative, reversible effect in exposed populations (NRC 1988). A health criterion for acute inhalation exposure to trichloroethene of  $100 \text{ mg/m}^3$  can be derived from the EEGL by combining it with a safety factor of 10 to account for the healthy worker effect, which assumes employed persons are healthier than the general population.

## REFERENCES

- American Conference of Governmental Industrial Hygienists (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th Ed. ACGIH, Cincinnati, Ohio.

- Environmental Protection Agency (EPA). 1985. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina. EPA/600/8-82/006F.
- Environmental Protection Agency (EPA). 1987. Health Advisory for Trichloroethylene. Office of Drinking Water, Washington, D.C. March 31, 1987.
- Environmental Criteria and Assessment Office (ECAO). 1992. Written correspondence from Joan Dollarhide, Chemical Mixtures Assessment Branch, U.S. Environmental Protection Agency. July 16, 1992.
- Fukuda, K., K. Takemoto, and H. Tsuruta. 1983. Inhalation Carcinogenicity of Trichloroethylene in Mice and Rats. *Ind. Health* 21:243-254.
- Greim, H., D. Bimboes, G. Egert, W. Giggelmann, and M. Kramer. 1977. Mutagenicity and Chromosomal Aberrations as an Analytical Tool for *in vitro* Detection of Mammalian Enzyme-Mediated Formation of Reactive Metabolites. *Arch. Toxicol.* 39:159.
- Kimmerle, G., and A. Eben. 1973. Metabolism, Excretion and Toxicology of Trichloroethylene After Inhalation. 1. Experimental Exposure on Rat. *Arch. Toxicol.* 30:115.
- Kjellstrand, P., B. Holmquist, N. Mandahl, and M. Bjerkemo. 1983a. Effects of Continuous Trichloroethylene Inhalation on Different Strains of Mice. *Acta Pharmacol. Toxicol.* 53:369-374.
- Kjellstrand, P., B. Holmquist, P. Alm, M. Kanje, S. Romare, I. Jonsson, L. Mansson, and M. Bjerkemo. 1983b. Trichloroethylene: Further Studies of the Effects on Body and Organ Weights and Plasma Butyrylcholinesterase Activity in Mice. *Acta Pharmacol. Toxicol.* 53:375-384.
- Maltoni, C., G. Lefemine, G. Cotti. 1986. Experimental Research on Trichloroethylene. Carcinogenesis *Arch. Res. Industrial Carcinogenesis Series*. C. Maltoni, M.A. Mehlman, Eds. Vol. V. Princeton Scientific Publishing Co., Inc., Princeton, New Jersey. p. 393.
- National Cancer Institute (NCI). 1976. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264 122.
- National Research Council (NRC). 1988. Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants. Volume 8. Prepared by the Committee on Toxicology. National Academy Press, 1988.
- National Toxicology Program (NTP). 1983. Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F<sub>1</sub> mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.
- Stephens, C. 1945. Poisoning by Accidental Drinking of Trichloroethylene. *Br. Med. J.* 2:218.
- Torkelson, T.R. and V.K. Rowe. 1981. Halogenated Aliphatic Hydrocarbons. In G.D. Clayton and P.B. Clayton, Eds. *Patty's Industrial Hygiene and Toxicology*. 3rd Ed. John Wiley and Sons, New York. Vol. 2B, pp. 3553-3559

## **AROCLOR 1254 (POLYCHLORINATED BIPHENYLS, PCBs)**

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

PCBs are readily and extensively absorbed through the gastrointestinal tract and somewhat less readily through the skin; PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (EPA 1985). Studies have found oral efficiency on the order of 75 to >90 percent in rats, monkeys and ferrets (Albro and Fishbein 1972; Allen et al. 1974; Tanabe et al. 1981; Bleavens et al. 1984; Clevenger et al. 1989). PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content (ATSDR 1991). Dermatitis and chloracne (a disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a study of women who had consumed PCB-contaminated fish from Lake Michigan (EPA 1985). Reproductive, hepatic, immunotoxic, and immunosuppressive effects appear to be the most sensitive end points of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents (EPA 1985). For example, adult monkeys exposed to dietary concentrations of 0.028 mg/kg-day Aroclor 1016 for approximately 22 months showed no evidence of overt toxicity; however, the offspring of these monkeys exhibited decreased birth weight and possible neurological impairment (Barsotti and Van Miller 1984; Levin et al. 1988; Schantz et al. 1989, 1991). A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors including liver tumors in animals exposed to the mixtures for long periods (Kimbrough et al. 1975; NCI 1978; Schaeffer et al. 1984; Norback and Weltman 1985). Studies have suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (EPA 1985). It is known that PCB congeners vary greatly in their potency in producing biological effects, such as cancer however, EPA (1995) generally considers Aroclor 1260 to be representative of all PCB mixtures for the evaluation of carcinogenic effects. There is some evidence that mixtures containing highly chlorinated biphenyls are more potent inducers of hepatocellular carcinoma in rats than are mixtures containing less chlorine by weight (EPA 1995).

EPA (1995) classified PCBs as a Group B2 agent (Probable Human Carcinogen) based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. The EPA (1995) calculated an oral cancer potency factor of  $7.7 \text{ (mg/kg/day)}^{-1}$  for PCBs based on the incidence of hepatocellular carcinomas (91 percent) and neoplastic nodules (4 percent) in female Sprague-Dawley rats exposed to a diet containing Aroclor 1260 as reported in a study by Norback and Weltman (1985). In the same study, males exhibited a much lower incidence of malignant tumors but a higher incidence of benign tumors (neoplastic nodules). EPA (1995) also calculated a slope factor of  $5.7 \text{ (mg/kg-day)}^{-1}$  for malignant tumors alone, which is supported by a risk estimate based on the data of Kimbrough et al. (1975). EPA (1995) derived an oral RfD of  $7 \times 10^{-5} \text{ mg/kg/day}$  for Aroclor 1016 based on a 21.8 month oral study conducted in monkeys (Barsotti and Van Miller 1984; Levin et al. 1988; Schantz et al. 1989, 1991). A no observed adverse effect level of 0.25 ppm (0.007 mg/kg/day) for decreased birth weight in offspring was identified from these studies. A safety factor of 100 (3 to account for interspecies extrapolation,

3 to account for sensitive individuals, 3 to account for limitations in the database, and 3 to account for extrapolation from a subchronic to a chronic RfD) was used to calculate the RfD.

EPA (1995) derived an oral RfD of  $2 \times 10^{-5}$  mg/kg/day for Aroclor 1254 based on clinical and immunological studies conducted in monkeys that received oral doses in gelatin capsules for more than five years (Arnold et al. 1994a,b; Tryphonas et al. 1989, 1991a,b; cited in EPA 1995). The critical effects observed in monkeys included ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails, and a decreased antibody (IgG and IgM) response to sheep erythrocytes. To derive the oral RfD, an uncertainty factor of 300 was applied to the LOAEL from the cited studies. The uncertainty factor accounts for the variability in toxic responses among human individuals, for the extrapolation of a study in monkeys to potential exposures of humans, and for the use of a LOAEL instead of a NOAEL.

## REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological Profile for Selected PCBs (Aroclor 1260, -1254, -1248, -1242, -1232, -1221, and 1016). U.S. Department of Health and Human Services. Draft for Public Comment. October 1991.
- Albro, P.W. and L. Fishbein. 1972. Intestinal Absorption of Polychlorinated Biphenyls in Rats. *Bull. Environ. Contam. Toxicol.* 8:26-31.
- Allen, J.R., D.H. Norback, and I.C. Hsu. 1974. Tissue Modifications in Monkeys as Related to Absorption, Distribution, and Excretion of Polychlorinated Biphenyls. *Arch. Environ. Contam. Toxicol.* 2:86-95.
- Barsotti, D.A. and J.P. Van Miller. 1984. Accumulation of a Commercial Polychlorinated Biphenyl Mixture (Aroclor 1016) in Adult Rhesus Monkeys and Their Nursing Infants. *Toxicology* 30:31-44.
- Bleavins, M.R., W.J. Breslin, and R.J. Aulerich et al. 1984. Placental and Mammary Transfer of a Polychlorinated Biphenyl Mixture (Aroclor 1254) in the European Ferret (*Mustela putorius furo*). *Environ. Toxicol. Chem.* 3:637-644.
- Clevenger, M.A., S.M. Roberts, and D.L. Lattin et al. 1989. The Pharmacokinetics of 2,2',5,5'-Tetrachlorobiphenyl and 3,3',4,4'-Tetrachlorobiphenyl and its Relationship to Toxicity. *Toxicol. Appl. Pharmacol.* 100:315-327.
- Environmental Protection Agency (EPA). 1985. Health Effects Criteria Document on Polychlorinated Biphenyls. Final Draft. Office of Drinking Water, Washington.
- Environmental Protection Agency (EPA). 1995. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Kimbrough, R.D., R.A. Squire, R.E. Linder, J.D. Strandberg, R.J. Montali, and V.W. Burse. 1975. Induction of Liver Tumors in Sherman Strain Female Rats by Polychlorinated Biphenyl Aroclor 1260. *J. Natl. Cancer Inst.* 55:1453.
- Levin, E.D., S.L. Schantz, and R.E. Bowman. 1988. Delayed Spatial Alteration Deficits Resulting from Perinatal PCB Exposure in Monkeys. *Arch. Toxicol.* 62:267-273.

National Cancer Institute (NCI). 1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. Cas. No. 27323-18-8. NCI Carcinogenesis Technical Report Series No. 38. DHEW (NIH) Publication No. 78-838.

Norback, D.H. and R.H. Weltman. 1985. Polychlorinated Biphenyl Induction of Hepatocellular Carcinoma in the Sprague-Dawley Rat. *Environ. Health Perspect.* 1:134-143.

Schaeffer, E., H. Greim, and W. Goessner. 1984. Pathology of Chronic Polychlorinated Biphenyl (PCB) Feeding in Rats. *Toxicol. Appl. Pharmacol.* 75:278-288.

Schantz, S.L., E.D. Levin, and R.E. Bowman et al. 1989. Effects of Perinatal PCB Exposure on Discrimination-Reversal Learning in Monkeys. *Neurotoxicol. Teratol.* 11:243-250.

Schantz, S.L., E.D. Levin, and R.E. Bowman. 1991. Long-Term Neurobehavioral Effects of Perinatal Polychlorinated Biphenyl (PCB) Exposure in Monkeys. *Environ. Toxicol. Chem.* 10:747-756.

Tanabe, S., Y. Nakagawa, and R. Tatsukawa. 1981. Absorption Efficiency and Biological Half-Life of Individual Chlorobiphenyls in Rats Treated with Kanechlor Products. *Agric. Biol. Chem.* 45:717-726..

## CARBON TETRACHLORIDE

Carbon tetrachloride is readily absorbed following oral and inhalation exposure. About 60 percent of an oral dose was absorbed within 6 hours, and up to 86 percent was absorbed by 24 hours. Absorption from the lung has been estimated at about 30 percent (EPA 1984). Carbon tetrachloride, like many other chlorinated hydrocarbons, acts as a CNS depressant (ACGIH 1986). The toxic effects of oral and inhalation exposure to carbon tetrachloride in humans and animals include damage to the liver, kidney and lung, although the liver is the most sensitive tissue (EPA 1985; Bruckner et al. 1986). In animals, acute oral administration produces fatty infiltration and histological alterations in the liver. High doses produce irreversible liver damage and necrosis, but the effects observed following lower doses are largely reversible (EPA 1985). Humans occupationally exposed to 5-15 ppm of carbon tetrachloride experience biochemical alterations, nausea, headaches and, in more severe cases, liver dysfunction (jaundice, enlargement and fatty infiltration) (ACGIH 1986; EPA 1984). Animals chronically exposed to carbon tetrachloride exhibit similar effects to those observed following acute exposure. Prenatal toxicity has been demonstrated in mammalian fetuses and neonates after inhalation exposure in pregnant rats (EPA 1984), although carbon tetrachloride has not been shown to be teratogenic (EPA 1985). Carbon tetrachloride is a carcinogen in animals producing mainly hepatic neoplasms. Oral administration of 30 mg/kg/day or higher for 6 months has been found to produce an increased frequency of hepatomas, hepatocellular adenomas and hepatocellular carcinomas in mice, rats and hamsters (EPA 1985).

EPA (1993) classified carbon tetrachloride as a B2 carcinogen (probable human carcinogen). The oral cancer slope factor is  $1.3 \times 10^{-1} \text{ (mg/kg/day)}^{-1}$  (EPA 1993) and is based on several gavage studies in which hepatocellular carcinomas and hepatomas were observed in rats, mice and hamsters (Della Porta et al. 1961; Edwards et al. 1942; NCI 1976a,b, 1977). EPA (1993) reported an inhalation cancer unit risk of  $1.5 \times 10^{-5} \text{ (}\mu\text{g/m}^3\text{)}^{-1}$ , based upon the same studies used to derive the oral slope factor. An absorption factor of 0.4 was used to calculate the inhalation unit risk from the oral slope factor to account for differences between oral and inhalation exposure. EPA (1993) derived an oral RfD of  $7 \times 10^{-4} \text{ mg/kg/day}$  based on a subchronic rat gavage study in which liver lesions were the most sensitive effect (Bruckner et al. 1986). A NOAEL of 0.71 mg/kg/day and an uncertainty factor of 1,000 were used to derive the RfD. EPA (1992) derived a subchronic oral RfD of  $7 \times 10^{-3} \text{ mg/kg/day}$ , using an uncertainty factor of 100, based on the same study and effect of concern.

## REFERENCES

- American Conference of Governmental Industrial Hygienists (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th Ed. ACGIH, Inc. Cincinnati, Ohio.
- Bruckner, J.V., W.F. Mackenzie, and S. Muralidhara et al. 1986. Oral Toxicity of Carbon Tetrachloride: Acute, Subacute and Chronic Studies in Rats. *Fund. Appl. Toxicol.* 6(1):16-34.
- Della Porta, G.B., B. Terracini, and K.P. Shubik. 1961. Induction with Carbon Tetrachloride of Liver Cell Carcinomas in Hamsters. *J. Natl. Cancer Inst.* 26:855-863.
- Edwards, J., W.E. Heston, and H.A. Dalton. 1942. Induction of Carbon Tetrachloride Hepatoma in Strain L Mice. *J. Natl. Cancer Inst.* 3:297-301.



Environmental Protection Agency (EPA). 1984. Health Assessment Document for Carbon Tetrachloride. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA-600/8-82-001F.

Environmental Protection Agency (EPA). 1985. Drinking Water Criteria Document for Carbon Tetrachloride (Final Draft). Office of Drinking Water, Washington, D.C. January 1985.

Environmental Protection Agency (EPA). 1992. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1992.

Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

National Cancer Institute (NCI). 1976a. Report on the Carcinogenesis Bioassay of Chloroform. Carcinogenesis Program, Division of Cancer Cause and Prevention, Washington, D.C.

National Cancer Institute (NCI). 1976b. Carcinogenesis Bioassay of Trichloroethylene. Carcinogenesis Technical Report Series No. 2, Washington, D.C.

National Cancer Institute (NCI). 1977. Bioassay of 1,1,1-Trichloroethane for Possible Carcinogenicity. Carcinogenesis Technical Report Series No. \*, Washington, D.C.

## CHLOROFORM

Chloroform, a trihalomethane, is rapidly absorbed through the respiratory and gastrointestinal tracts in humans and experimental animals; dermal absorption from contact of the skin with liquid chloroform can also occur (EPA 1985). In humans, acute exposures to chloroform may result in depression of the CNS, hepatic and renal damage, and death caused by ventricular fibrillation following an acute ingested dose of 10 ml (EPA 1984). Acute exposure to chloroform may also cause irritation to the skin, eyes, and gastrointestinal tract (EPA 1984, 1985). In experimental animals, chronic exposure may lead to fatty cyst formation in the liver (Heywood et al. 1979), renal and cardiac effects, and CNS depression (EPA 1985). Chloroform has been reported to induce renal epithelial tumors in rats (Jorgenson et al. 1985) and hepatocellular carcinomas in mice (NCI 1976). Suggestive evidence from human epidemiological studies indicates that long-term exposure to chloroform and other trihalomethanes in contaminated water supplies may be associated with an increased incidence of bladder tumors (EPA 1985). Chloroform is embryotoxic in pregnant rats and has retarded fetal development and increased the incidences of fetal resorption, absence of tail, imperforate anus, missing ribs and delayed ossification of sternebrae (Schwetz et al. 1974).

Chloroform has been classified by EPA (1993) as a Group B2 Carcinogen (Probable Human Carcinogen). EPA (1993) developed an oral cancer slope factor for chloroform of  $6.1 \times 10^{-3}$  (mg/kg/day)<sup>-1</sup> based on a study in which kidney tumors were observed in rats exposed to chloroform in drinking water (Jorgenson et al. 1985). An inhalation cancer unit risk of  $2.3 \times 10^{-5}$  (μg/m<sup>3</sup>)<sup>-1</sup> has been developed by EPA (1993) based on an NCI (1976) bioassay in which liver tumors were observed in mice. EPA also derived an oral RfD of  $1 \times 10^{-2}$  mg/kg/day for both chronic (EPA 1993) and subchronic (EPA 1992) exposure to chloroform, based on a chronic bioassay in dogs in which liver effects were observed at 12.9 mg/kg/day (Heywood et al. 1979); an uncertainty factor of 1,000 was used to derive the RfD for both exposure durations.

The National Research Council's Committee on Toxicology has set a one-hour EEGL, for chloroform at 100 ppm (500 mg/m<sup>3</sup>) (NRC 1984). Formerly known as EEL, the EEGL is defined as a ceiling limit for an unpredicted single exposure lasting one to 24 hours, which is expected to be rare in the lifetime of any person. It is designed to avoid substantial decrements in performance during emergencies and takes into account the statistical likelihood of a non-incapacitating, reversible effect in exposed populations (NRC 1984). A health criterion for acute inhalation exposure to chloroform of 50 mg/m<sup>3</sup> can be derived from the EEGL by combining it with a safety factor of 10 to account for the healthy worker effect, which assumes employed persons are healthier than the general population.

## REFERENCES

- Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Chloroform. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-010.
- Environmental Protection Agency (EPA). 1985. Health Assessment Document for Chloroform. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. September 1985. EPA 600/8-84-004F.

- Environmental Protection Agency (EPA). 1992. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1992.
- Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.
- Heywood, R., R.J. Sortwell, and P.R.B. Noel et al. 1979. Safety Evaluation of Toothpaste Containing Chloroform. III. Long-Term Study in Beagle Dogs. J. Environ. Pathol. Toxicol. 2:835-851.
- Jorgenson, T.A., E.F. Meierhenry, and C.J. Rushbrook et al. 1985. Carcinogenicity of Chloroform in Drinking Water to Male Osborne-Mendel Rats and Female B6C3F1 Mice. Fund. Appl. Toxicol. 5:760-769.
- National Cancer Institute (NCI). 1976. Carcinogenesis Bioassay of Chloroform. CAS No. 67-66-3. NCI Carcinogenesis Technical Report Series No. 0. Bethesda, M.D. DHEW (NIH) Publication No. 76.
- National Research Council (NRC). 1984. Emergency and Continuous Exposure Limits for Selected Airborne Contaminants. Volume 1. Prepared by the Committee on Toxicology. National Academy Press, April 1984.
- Schwetz, B.A., B.K.J. Leong, and P.J. Gehring. 1974. Embryo- and Fetotoxicity of Inhaled Chloroform in Rats. Toxicol. Appl. Pharmacol. 28:442-451.

**APPENDIX C**

**CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS**

## CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION Cape Lisburne	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
SPECIES brown lemming				
COC				
<b>Inorganics</b>				
Iron	0.00	0.004	1.00	0.000
Manganese	0.00	0.25	1.00	0.000
<b>Organics</b>				
DRPH	4464	0.03	1.00	149.391
Benzene	0.08	2.27	1.00	0.182
Toluene	0.43	1.08	1.00	0.464
Xylenes (total)	0.82	0.97	1.00	0.796
1,4-Dichlorobenzene	0.06	0.43	1.00	0.026
4-Methylphenol	3.84	2.97	1.00	11.397
1,2,4-Trimethylbenzene	0.00	0.25	1.00	0.000
Aroclor 1254	27.96	0.004	1.00	0.105
Aroclor 1260	273.77	0.004	1.00	1.075

## CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION	COC	Bioconcentration	Proportion	COC
Cape Lisburne	Concentration	Factor	of vegetation	Concentration
SPECIES	Soil/Sediment		in diet	in food
Lapland longspur	(CS)	(Bv)	(%V)	(CF)
COC	mg/kg	unitless	%	mg/kg
<b>Inorganics</b>				
Iron	0.00	0.004	0.25	0.000
Manganese	0.00	0.25	0.25	0.000
<b>Organics</b>				
DRPH	4464	0.03	0.25	37.348
Benzene	0.08	2.27	0.25	0.045
Toluene	0.43	1.08	0.25	0.116
Xylenes (total)	0.82	0.97	0.25	0.199
1,4-Dichlorobenzene	0.06	0.43	0.25	0.006
4-Methylphenol	3.84	2.97	0.25	2.849
1,2,4-Trimethylbenzene	0.00	0.25	0.25	0.000
Aroclor 1254	27.96	0.004	0.25	0.026
Aroclor 1260	273.77	0.004	0.25	0.269

## CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION Cape Lisburne	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
SPECIES brant				
COC				
<b>Inorganics</b>				
Iron	0.00	0.004	0.90	0.000
Manganese	0.00	0.250	0.90	0.000
<b>Organics</b>				
DRPH	4464	0.03	0.90	134.452
Benzene	0.08	2.27	0.90	0.164
Toluene	0.43	1.08	0.90	0.418
Xylenes (total)	0.82	0.97	0.90	0.716
1,4-Dichlorobenzene	0.06	0.43	0.90	0.023
4-Methylphenol	3.84	2.97	0.90	10.257
1,2,4-Trimethylbenzene	0.00	0.25	0.90	0.000
Aroclor 1254	27.96	0.004	0.90	0.095
Aroclor 1260	273.77	0.004	0.90	0.967

## CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS				
CF = CS*Bv*%V				
INSTALLATION Cape Lisburne	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
SPECIES glaucous gull				
COC				
<b>Inorganics</b>				
Iron	0.00	0.004	0.10	0.000
Manganese	0.00	0.25	0.10	0.000
<b>Organics</b>				
DRPH	4464	0.03	0.10	14.939
Benzene	0.08	2.27	0.10	0.018
Toluene	0.43	1.08	0.10	0.046
Xylenes (total)	0.82	0.97	0.10	0.080
1,4-Dichlorobenzene	0.06	0.43	0.10	0.003
4-Methylphenol	3.84	2.97	0.10	1.140
1,2,4-Trimethylbenzene	0.00	0.25	0.10	0.000
Aroclor 1254	27.96	0.004	0.10	0.011
Aroclor 1260	273.77	0.004	0.10	0.107



## **APPENDIX D**

### **ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS**

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001}{IS}$ / BW																
INSTALLATION	COC Conc.	Food Intake Rate	Soil/Sed. Intake %	COC Conc.	Soil/Sed. Ingestion Rate	Relative Oral Availability	COC Conc.	Water Intake Rate	Percent Ingested at Site	Conver. Units	(A+B+C)	(D)	(D)*.001	(unitless)	Body Weight (BW)	ESTIMATED EXPOSURE (D*IS/BW*EE)
SPECIES	Food Items (CF)*	(FI)	(CF*FI)	(SI%)	(CS)	(SI)	(ROA)	(CS*SI*ROA)	(CW)	(WI)	(CW*WI)	(A+B+C)	(D)	(D)*.001	(IS)	(BW)
	(mg/kg)	(g/day)	(A)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(ug/L)	(L/day)	(C)	(D)	(D)*.001	(unitless)	(kg)	(mg/kg-bw/day)
Inorganics																
Iron	0.0000	45	0.00	0.027	0.00	1.22	1	0.00	2405.00	0.007	16.835	16.84	0.02	0.5	0.055	0.1530
Manganese	0.0000	45	0.00	0.027	0.00	1.22	1	0.00	670.00	0.007	4.69	4.69	0.00	0.5	0.055	0.0426
Organics																
DRPH	149.3909	45	6722.59	0.027	4464.00	1.22	1	5423.76	0.00	0.007	0	12146.35	12.15	0.5	0.055	110.4214
Benzene	0.1819	45	8.19	0.027	0.08	1.22	1	0.10	0.00	0.007	0	8.28	0.01	0.5	0.055	0.0753
Toluene	0.4641	45	20.89	0.027	0.43	1.22	1	0.52	0.00	0.007	0	21.41	0.02	0.5	0.055	0.1946
Xylenes (total)	0.7957	45	35.81	0.027	0.82	1.22	1	1.00	0.00	0.007	0	36.80	0.04	0.5	0.055	0.3346
1,4-Dichlorobenzene	0.0255	45	1.15	0.027	0.06	1.22	1	0.07	0.00	0.007	0	1.22	0.00	0.5	0.055	0.0111
4-Methylphenol	11.3970	45	512.87	0.027	3.84	1.22	1	4.67	0.00	0.007	0	517.53	0.52	0.5	0.055	4.7048
1,2,4-Trimethylbenzene	0.0000	45	0.00	0.027	0.00	1.22	1	0.00	0.61	0.007	0.00427	0.00	0.00	0.5	0.055	0.0000
Aroclor 1254	0.1055	45	4.75	0.027	27.96	1.22	1	33.97	0.00	0.007	0	38.72	0.04	0.5	0.055	0.3520
Aroclor 1260	1.0750	45	48.37	0.027	273.77	1.22	1	332.63	0.00	0.007	0	381.01	0.38	0.5	0.055	3.4637

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001}{IS} / BW$														
INSTALLATION	COC Conc.	Food Intake Rate (FI)	Soil/Sed. Intake % (SI%)	Soil/Sed. Ingestion Rate (SI)	COC Conc. Soil/Sed. (CS)	Relative Oral Availability (ROA)	COC Conc. Water (CW)	Water Intake Rate (WI)	(C) (CW*WI)	(A+B+C)	Conver. Units 0.0010	Percent Ingested at Site (IS)	Body Weight (BW)	ESTIMATED EXPOSURE (D*IS/BW=EE)
SPECIES	Food Items (CF)*	(FI)	(SI%)	(SI)	(CS)	(ROA)	(SI*CS*ROA)	(WI)	(C)	(D)	(D)*.001	(unitless)	(kg)	(mg/kg-bw/day)
COC	(mg/kg)	(g/day)	(A)	% of FI	(g/day)	(unitless)	(B)	(ug/L)	(L/day)	(D)	(D)*.001	(unitless)	(kg)	(mg/kg-bw/day)
Inorganics														
Iron		256	0	0.028	7.17	1	0.00	2405.00	0.42	1010.1	1.01	0.03	4.95	0.0061
Manganese		256	0	0.028	7.17	1	0.00	670.00	0.42	281.4	0.28	0.03	4.95	0.0017
Organics														
DRPH		256	0	0.028	7.17	1	31997.95	0.00	0.42	0	31997.95	0.03	4.95	0.1939
Benzene		256	0	0.028	7.17	1	0.57	0.00	0.42	0	0.57	0.03	4.95	0.0000
Toluene		256	0	0.028	7.17	1	3.08	0.00	0.42	0	3.08	0.03	4.95	0.0000
Xylenes (total)		256	0	0.028	7.17	1	5.88	0.00	0.42	0	5.88	0.03	4.95	0.0000
1,4-Dichlorobenzene		256	0	0.028	7.17	1	0.43	0.00	0.42	0	0.43	0.03	4.95	0.0000
4-Methylphenol		256	0	0.028	7.17	1	27.53	0.00	0.42	0	27.53	0.03	4.95	0.0002
1,2,4-Trimethylbenzene		256	0	0.028	7.17	1	0.00	0.61	0.42	0.2562	0.26	0.03	4.95	0.0000
Aroclor 1254		256	0	0.028	7.17	1	200.42	0.00	0.42	0	200.42	0.20	4.95	0.0012
Aroclor 1260		256	0	0.028	7.17	1	1962.38	0.00	0.42	0	1962.38	1.96	4.95	0.0119

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * .001\} * IS\} / BW$												
INSTALLATION	COC	Food	Soil/Sed.	COC	Soil/Sed.	Relative	COC	Water	Conver.	Percent	Body	ESTIMATED
Cape Lisburne	Conc.	Intake	Intake	Conc.	Intake	Oral	Conc.	Intake	Units	at Site	Weight	EXPOSURE
SPECIES	Food Items	Rate	%	Soil / Sed.	%	Availability	Water	Rate	(A+B+C)	(IS)	(BW)	(D*IS/BW=EE)
caribou	(CF*FI)	(FI)	(SI%)	(CS)	(SI)	(ROA)	(CW)	(WI)				
COC	(mg/kg)	(g/day)	(A)	(mg/kg)	(g/day)	(unitless)	(ug/L)	(L/day)	(D)	(D)*.001	(kg)	(mg/kg-bw/day)
Inorganics												
Iron		2400	0.00	0.00	48	1	2405.00	6.0	14430.00	14.43	95.5	0.0015
Manganese	0.0000	2400	0.00	0.00	48	1	670.00	6.0	4020.00	4.02	95.5	0.0004
Organics												
DRPH	149.3909	2400	358538.19	4464.00	48	1	214272.00	6.0	0	572810.19	95.5	0.0600
Benzene	0.1819	2400	436.68	0.08	48	1	3.84	6.0	0	440.52	95.5	0.0000
Toluene	0.4641	2400	1113.93	0.43	48	1	20.64	6.0	0	1134.57	95.5	0.0001
Xylenes (total)	0.7957	2400	1909.70	0.82	48	1	39.36	6.0	0	1949.06	95.5	0.0002
1,4-Dichlorobenzene	0.0255	2400	61.23	0.06	48	1	2.88	6.0	0	64.11	95.5	0.0000
4-Methylphenol	11.3970	2400	27352.84	3.84	48	1	184.32	6.0	0	27537.16	95.5	0.0029
1,2,4-Trimethylbenzene	0.0000	2400	0.00	0.00	48	1	0.00	6.0	3.66	0.00	95.5	0.0000
Aroclor 1254	0.1055	2400	253.18	27.96	48	1	1342.08	6.0	0	1595.26	95.5	0.0002
Aroclor 1260	1.0750	2400	2579.98	273.77	48	1	13140.96	6.0	0	15720.94	95.5	0.0016

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001 * IS}{BW}$																			
INSTALLATION	COC	Food Intake	Soil/Sed. Intake	COC Conc.	Soil/Sed. Conc.	Relative Oral Availability	COC Conc.	Water Intake Rate	(CW*WI)	(A+B+C)	Conver. Units	Percent Ingested at Site	Body Weight	ESTIMATED EXPOSURE					
SPECIES	Food Items (CF)*	Rate (FI)	(CF*FI)	(SI%)	(CS)	(SI)	(CS*SI*ROA)	(WI)	(CW*WI)		0.0010	(IS)	(BW)	(D'IS/BW=EE)					
Lapland longspur																			
COC	(mg/kg)	(g/day)	(A)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(ug/L)	(C)	(D)	(D)*.001	(unitless)	(kg)	(mg/kg-bw/day)				
Inorganics																			
Iron		6.6	0.00	0.02	0.00	0.13	1	0.00	2405.00	0.005	12.03	0.01	1.0	0.027	0.4454				
Manganese	0.0000	6.6	0.00	0.02	0.00	0.13	1	0.00	670.00	0.005	3.35	0.00	1.0	0.027	0.1241				
Organics																			
DRPH	37.3477	6.6	246.50	0.02	4464.00	0.13	1	589.25	0.00	0.005	0.00	835.74	1.0	0.027	30.9534				
Benzene	0.0455	6.6	0.30	0.02	0.08	0.13	1	0.01	0.00	0.005	0.00	0.31	1.0	0.027	0.0115				
Toluene	0.1160	6.6	0.77	0.02	0.43	0.13	1	0.06	0.00	0.005	0.00	0.82	1.0	0.027	0.0305				
Xylenes (total)	0.1989	6.6	1.31	0.02	0.82	0.13	1	0.11	0.00	0.005	0.00	1.42	1.0	0.027	0.0526				
1,4-Dichlorobenzene	0.0064	6.6	0.04	0.02	0.06	0.13	1	0.01	0.00	0.005	0.00	0.05	1.0	0.027	0.0019				
4-Methylphenol	2.8493	6.6	18.81	0.02	3.84	0.13	1	0.51	0.00	0.005	0.00	19.31	1.0	0.027	0.7153				
1,2,4-Trimethylbenzene	0.0000	6.6	0.00	0.02	0.00	0.13	1	0.00	0.61	0.005	0.00	0.00	1.0	0.027	0.0001				
Aroclor 1254	0.0264	6.6	0.17	0.02	27.96	0.13	1	3.69	0.00	0.005	0.00	3.86	1.0	0.027	0.1431				
Aroclor 1260	0.2687	6.6	1.77	0.02	273.77	0.13	1	36.14	0.00	0.005	0.00	37.91	1.0	0.027	1.4041				

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\frac{((CF*FI) + (CS*SI*ROA) + (CW*WI)) * .001 * IS}{BW}$														
INSTALLATION	COC Conc.	Food Intake Rate (FI)	Food Intake Rate (FI)	Soil/Sed. Intake % (SI%)	COC Conc. (CS)	Soil/Sed. Ingestion Rate (SI)	Relative Oral Availability (ROA)	COC Conc. (CS)	Water Intake Rate (WI)	COC Conc. (CW)	Water Intake Rate (WI)	Conver. Units (D)*.001	Percent Ingested at Site (IS)	ESTIMATED EXPOSURE (D*IS/BW=EE)
SPECIES	Food Items (CF)*	(FI)	(FI)	(SI%)	(CS)	(SI)	(ROA)	(CS)	(WI)	(CW)	(WI)	0.0010	(IS)	(D*IS/BW=EE)
glaucous gull														
COC	(mg/kg)	(g/day)	(g/day)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(L/day)	(ug/L)	(L/day)	(D)	(unitless)	(mg/kg-bw/day)
Inorganics														
Iron		73.9	73.9	0.00	0.00	5.62	1	0.00	0.08	2405.00	0.08	192.40	0.19	0.0119
Manganese	0.0000	73.9	73.9	0.00	0.00	5.62	1	0.00	0.08	670.00	0.08	53.60	0.05	0.0033
Organics														
DRPH	14.9391	73.9	73.9	1104.00	4464.00	5.62	1	25071.61	0.08	0.00	0.08	26175.61	0.09	1.6247
Benzene	0.0182	73.9	73.9	1.34	0.08	5.62	1	0.45	0.08	0.00	0.08	1.79	0.00	0.0001
Toluene	0.0464	73.9	73.9	3.43	0.43	5.62	1	2.42	0.08	0.00	0.08	5.85	0.01	0.0004
Xylenes (total)	0.0796	73.9	73.9	5.88	0.82	5.62	1	4.61	0.08	0.00	0.08	10.49	0.01	0.0007
1,4-Dichlorobenzene	0.0026	73.9	73.9	0.19	0.06	5.62	1	0.34	0.08	0.00	0.08	0.53	0.00	0.0000
4-Methylphenol	1.1397	73.9	73.9	84.22	3.84	5.62	1	21.57	0.08	0.00	0.08	105.79	0.11	0.0066
1,2,4-Trimethylbenzene	0.0000	73.9	73.9	0.00	0.00	5.62	1	0.00	0.08	0.61	0.08	0.05	0.00	0.0000
Aroclor 1254	0.0105	73.9	73.9	0.78	27.96	5.62	1	157.03	0.08	0.00	0.08	157.81	0.16	0.0098
Aroclor 1260	0.1075	73.9	73.9	7.94	273.77	5.62	1	1537.60	0.08	0.00	0.08	1545.55	1.55	0.0959

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = $\{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * 0.01\} * IS\} / BW$																	
INSTALLATION Cape Lisburne	SPECIES brant	COC Conc. Food Items (CF)*	Food Intake Rate (FI)	COC Conc. Soil/Sed. (CS)	Soil/Sed. Intake % (SI%)	COC Conc. Soil/Sed. (CS)	Soil/Sed. Ingestion Rate (SI)	Relative Oral Availability (ROA)	COC Conc. Water (CW)	Water Intake Rate (WI)	(C)	(D)	Conver. Units 0.0010	Percent Ingested at Site (IS)	Body Weight (BW)	ESTIMATED EXPOSURE (D*IS/BW*EE)	
																	(mg/kg)
Inorganics																	
Iron			69.2	0.00	0.082	0.00	5.67	1	0.00	2405.00	0.07	168.35	168.35	0.17	0.56	1.31	0.0720
Manganese		0.0000	69.2	0.00	0.082	0.00	5.67	1	0.00	670.00	0.07	46.9	46.90	0.05	0.56	1.31	0.0200
Organics																	
DRPH		134.4518	69.2	9304.07	0.082	4464.00	5.67	1	25330.52	0.00	0.07	0	34634.59	34.63	0.56	1.31	14.8056
Benzene		0.1638	69.2	11.33	0.082	0.08	5.67	1	0.45	0.00	0.07	0	11.79	0.01	0.56	1.31	0.0050
Toluene		0.4177	69.2	28.91	0.082	0.43	5.67	1	2.44	0.00	0.07	0	31.35	0.03	0.56	1.31	0.0134
Xylenes (total)		0.7161	69.2	49.56	0.082	0.82	5.67	1	4.65	0.00	0.07	0	54.21	0.05	0.56	1.31	0.0232
1,4-Dichlorobenzene		0.0230	69.2	1.59	0.082	0.06	5.67	1	0.34	0.00	0.07	0	1.93	0.00	0.56	1.31	0.0008
4-Methylphenol		10.2573	69.2	709.81	0.082	3.84	5.67	1	21.79	0.00	0.07	0	731.60	0.73	0.56	1.31	0.3127
1,2,4-Trimethylbenzene		0.0000	69.2	0.00	0.082	0.00	5.67	1	0.00	0.61	0.07	0.0427	0.04	0.00	0.56	1.31	0.0000
Aroclor 1254		0.0949	69.2	6.57	0.082	27.96	5.67	1	158.66	0.00	0.07	0	165.23	0.17	0.56	1.31	0.0706
Aroclor 1260		0.9675	69.2	66.9506	0.082	273.77	5.67	1	1553.48	0.00	0.07	0	1620.43	1.62	0.56	1.31	0.6927

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.

# ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

$$\text{Estimated Exposure} = \{[(CF*FI) + (CS*SI*ROA) + (CW*WI)] * 0.001\} * IS / BW$$

INSTALLATION		COC															
Cape Lisburne		COC Conc.	Food Intake	Soil/Sed. Intake	COC Conc.	Soil/Sed. Ingestion	Relative Oral	COC Conc.	Water Intake	Percent Ingested	Body Weight	ESTIMATED EXPOSURE					
SPECIES		Food Items (CF)*	Rate (FI)	(CF*FI)	(SI%)	Soil/Sed. (CS)	Rate (SI)	(ROA)	(CS*SI*ROA)	(CW)	(CW*WI)	(A+B+C)	Units 0.0010	(IS)	(BW)	(D*IS/BW=EE)	
pectoral sandpiper		(mg/kg)	(g/day)	(A)	% of FI	(mg/kg)	(g/day)	(unitless)	(B)	(ug/L)	(L/day)	(C)	(D)	(D)*.001 (unitless)	(kg)	(mg/kg-bw/day)	
Inorganics	Iron		11.1	0.0000	0.181	0.00	2.01	1	0.00	2405.00	0.01	24.05	24.05	0.02	1.0	0.08	0.3006
	Manganese	0.0000	11.1	0.0000	0.181	0.00	2.01	1	0.00	670.00	0.01	6.7	6.70	0.01	1.0	0.08	0.0838
Organics	DRPH	14.9391	11.1	165.8239	0.181	4464.00	2.01	1	8968.62	0.00	0.01	0	9134.45	9.13	1.0	0.08	114.1806
	Benzene	0.0182	11.1	0.2020	0.181	0.08	2.01	1	0.16	0.00	0.01	0	0.36	0.00	1.0	0.08	0.0045
	Toluene	0.0464	11.1	0.5152	0.181	0.43	2.01	1	0.86	0.00	0.01	0	1.38	0.00	1.0	0.08	0.0172
	Xylenes (total)	0.0796	11.1	0.8932	0.181	0.82	2.01	1	1.65	0.00	0.01	0	2.53	0.00	1.0	0.08	0.0316
	1,4-Dichlorobenzene	0.0026	11.1	0.0283	0.181	0.06	2.01	1	0.12	0.00	0.01	0	0.15	0.00	1.0	0.08	0.0019
	4-Methylphenol	1.1397	11.1	12.6507	0.181	3.84	2.01	1	7.71	0.00	0.01	0	20.37	0.02	1.0	0.08	0.2546
	1,2,4-Trimethylbenzene	0.0000	11.1	0.0000	0.181	0.00	2.01	1	0.00	0.61	0.01	0.0061	0.01	0.00	1.0	0.08	0.0001
	Aroclor 1254	0.0105	11.1	0.1171	0.181	27.96	2.01	1	56.17	0.00	0.01	0	56.29	0.06	1.0	0.08	0.7036
	Aroclor 1260	0.0464	11.1	0.5152	0.181	273.77	2.01	1	550.03	0.00	0.01	0	550.55	0.55	1.0	0.08	6.8818

\*Concentration in Food cannot be calculated for the arctic fox because COC data is not available for animal portion of trophic web.



**APPENDIX E**

**BIOCONCENTRATION FACTOR CALCULATIONS  
FOR ORGANIC CHEMICALS**

# BIOCONCENTRATION FACTOR CALCULATIONS for ORGANIC CHEMICALS

CALCULATION OF Bv FOR ORGANIC CHEMICALS IN SOIL				
COC Organics	log Kow	$1.588 - 0.578 \log Kow$	log Bv	Bv
DRPH	5.30	-1.475	-1.475	0.033
Benzene	2.13	0.357	0.357	2.274
Toluene	2.69	0.033	0.033	1.079
Xylenes (total)	2.77	-0.013	-0.013	0.970
1,4-Dichlorobenzene	3.39	-0.371	-0.371	0.425
4-Methylphenol	1.93	0.472	0.472	2.968
Aroclor 1254	6.94	-2.423	-2.423	0.004
Aroclor 1260	6.91	-2.406	-2.406	0.004

\* log Kow for ortho-xylene used

**APPENDIX F**  
**SCALING FACTOR CALCULATIONS**

## SCALING FACTOR CALCULATIONS

Scaling factor (SF) = (representative species average body weight/ test species average body weight)<sup>1/3</sup>  
 based on the mass to surface area ratios of the test species and the representative species  
 (Mantel and Schneiderman 1975)

Representative Species	Average Body Weight <sup>1</sup> (kg)	Test Species	Average Body Weight <sup>2</sup> (kg)	Scaling Factor (SF)
brown lemming	0.055	mouse	0.025	1.30
	0.055	rat	0.25	0.60
arctic fox	4.95	rat	0.25	2.70
	4.95	mouse	0.025	5.82
	4.95	mink	1.0	1.70
caribou	95.5	sheep	60	1.17
	95.5	cattle	500	0.58
	95.5	rat	0.25	7.24
	95.5	mouse	0.025	15.59
Lapland longspur	0.027	chicken	0.8	0.32
	0.027	mallard	1.08	0.29
	0.027	Japanese quail	0.10	0.65
	0.027	ringed dove	0.155	0.56
	0.027	ring-necked pheasant	1.140	0.29
brant	1.305	chicken	0.8	1.18
	1.305	mallard	1.08	1.07
	1.305	Japanese quail	0.10	2.35
	1.305	ringed dove	0.155	2.03
	1.305	ring-necked pheasant	1.140	1.05
glaucous gull	1.445	chicken	0.8	1.22
	1.445	mallard	1.08	1.10
	1.445	Japanese quail	0.10	2.43
	1.445	ringed dove	0.155	2.10
	1.445	ring-necked pheasant	1.140	1.08
pectoral sandpiper	0.079	chicken	0.8	0.46
	0.079	mallard	1.08	0.42
	0.079	Japanese quail	0.10	0.92
	0.079	ringed dove	0.155	0.80
	0.079	ring-necked pheasant	1.140	0.41

## **APPENDIX G**

### **DATA SUMMARY TABLES**

G-1.	ALL (SUMMARY OF RI SAMPLING AND ANALYSIS) .....	G-1
G-2.	BACKGROUND ANALYTICAL DATA SUMMARY .....	G-3
G-3.	LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY .	G-12
G-4.	WHITE ALICE SITE ANALYTICAL DATA SUMMARY .....	G-34
G-5.	SPILL/LEAK #3 ANALYTICAL DATA SUMMARY .....	G-37
G-6.	UPPER CAMP TRANSFORMER BUILDING ANALYTICAL DATA SUMMARY .....	G-48
G-7.	LOWER CAMP TRANSFORMER BUILDINGS ANALYTICAL DATA SUMMARY .....	G-50
G-8.	WATER GALLERY ANALYTICAL DATA SUMMARY .....	G-53

**TABLE G-1. SUMMARY OF SAMPLING AND ANALYSES CONDUCTED**

ANALYSES	HVOC*	BTEX*	VOC 8260	SVOC	Metals <sup>b</sup>	TPH-Diesel <sup>b</sup> Range 3510/3550
ANALYTICAL METHOD	SW8010M	SW8020	SW8260	SW8270	SW3050 (Soil) 3005 (Water)/6010	Diesel 8100M
CAPE LISBON						
Background (BKGD)	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	3 Soil 2 Water	5 Soil 2 Water (Total) 2 Water (Dissolved)	5 Soil 2 Water
Landfill Waste Accumulation Area (LF01)	13 Soil	13 Soil 6 Water	12 Soil 5 Water	11 Soil 6 Water	4 Soil 4 Water (Total) 4 Water (Dissolved)	19 Soil 8 Water
White Alice Site (SS03)	NA	NA	NA	NA	NA	11 Soil
Spill/Leak #3 (ST07)	NA	27 Soil 1 Water	9 Soil 2 Water	1 Soil 1 Water	NA	36 Soil 2 Water
Upper Camp Transformer Building (SS08)	NA	NA	NA	NA	NA	7 Soil
Lower Camp Transformer Buildings (SS09)	NA	NA	NA	NA	NA	10 Soil
Water Gallery (AOC3)	NA	NA	5 Water	5 Water	5 Water (Total) 4 Water (Dissolved)	1 Water
Total Field Analyses	18 Soil 2 Water	45 Soil 9 Water	40 Soil 14 Water	15 Soil 14 Water	9 Soil 11 Water (Total) 10 Water (Dissolved)	88 Soil 13 Water
QA/QC SAMPLES						
Trip Blanks	2 Water	4 Water	9 Water	NA	NA	NA
Equipment Blanks	2 Water	4 Water	5 Water	2 Water	2 Water (Total) 1 Water (Dissolved)	5 Water
Ambient Condition Blanks	NA	NA	2 Water	NA	NA	NA
Field Replicates	2 Soil	3 Soil	5 Soil	3 Soil	2 Soil	7 Soil
Field Duplicates	NA	2 Water	3 Water	2 Water	1 Water (Total) 1 Water (Dissolved)	3 Water
Total Site Analyses	20 Soil 6 Water	48 Soil 19 Water	45 Soil 33 Water	18 Soil 18 Water	11 Soil 14 Water (Total) 12 Water (Dissolved)	95 Soil 21 Water

NA Not analyzed.

\* These analyses were completed on a quick turnaround basis.

<sup>a</sup> The number of soil sample includes sediment samples collected from surface water features.

<sup>b</sup> Some of these analysis were completed on a 24-hour turnaround at a temporary fixed laboratory at Barrow.

ES CONDUCTED FOR CAPE LISBURNE REMEDIAL INVESTIGATIONS\*

TPH-Diesel <sup>a</sup> Range 3510/3550	TPH - Gasoline <sup>b</sup> Range	TPH Residual Range*	PCB*	Pesticides*	TDS	TSS	TOC	TOTAL SAMPLES
Diesel 8100M	Gas 5030/8015M	Diesel 8100M	SW8080/8080M	SW8080/8080M	E160.1	E160.2	SW9060	
CAPE LISBURNE								
5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	5 Soil 2 Water	2 Water	2 Water	2 Soil 2 Water	12 Soil 2 Water
19 Soil 8 Water	22 Soil 6 Water	19 Soil 8 Water	41 Soil 6 Water	1 Soil 1 Water	4 Water	4 Water	4 Water	47 Soil 8 Water
11 Soil	NA	11 Soil	16 Soil	1 Soil	NA	NA	NA	16 Soil
36 Soil 2 Water	16 Soil 1 Water	26 Soil 2 Water	NA	1 Soil 1 Water	2 Water	2 Water	1 Water	36 Soil 2 Water
7 Soil	NA	7 Soil	8 Soil	NA	NA	NA	1 Soil	8 Soil
10 Soil	NA	10 Soil	13 Soil	1 Soil	NA	NA	NA	13 Soil
1 Water	NA	1 Water	2 Water	5 Water	4 Water	4 Water	4 Water	5 Water
88 Soil 13 Water	43 Soil 9 Water	78 Soil 13 Water	90 Soil 10 Water	9 Soil 9 Water	12 Water	12 Water	2 Soil 11 Water	132 Soil 17 Water
NA	3 Water	NA	NA	NA	NA	NA	NA	10 Water
5 Water	4 Water	4 Water	4 Water	3 Water	NA	NA	NA	9 Water
NA	NA	NA	3 Water	NA	NA	NA	NA	2 Water
7 Soil	4 Soil	7 Soil	5 Soil	1 Soil	NA	NA	1 Soil	8 Soil
3 Water	2 Water	3 Water	1 Water	1 Water	2 Water	2 Water	2 Water	3 Water
95 Soil 21 Water	47 Soil 18 Water	85 Soil 20 Water	95 Soil 18 Water	10 Soil 13 Water	14 Water	14 Water	3 Soil 13 Water	140 Soil 41 Water

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY

Installation: Cape Lisburne Site: Background (BKGD)		Matrix: Soil/Sediment Units: mg/kg										Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples					Field Blanks			
					S01	S02	S03	S04	SD01	AB01	EB01	TB01	
Laboratory Sample ID Numbers					1382/1554 4477-1 4476-2	1555/1556 4481-4	1574 4481-5	1576 4481-6	1381/1557 4477-2 4476-3	4512-3	1558 1561 4476-5	1552 4476-1	#182-9493 #6-9593 #6-9293 4476/4512 4481/4476/4477
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/L	mg/L	mg/kg
DRPH	6-15	60-150	500 <sup>a</sup>	<80 <sup>b</sup> <150 <sup>b</sup>	<130 <sup>b</sup>	<60 <sup>b</sup>	<120 <sup>b</sup>	<150 <sup>b</sup>	<80 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<50
GRPH	0.2-0.6	2-6	100	<20 <sup>b</sup> <50 <sup>b</sup>	<40 <sup>b</sup>	<20 <sup>b</sup>	<40 <sup>b</sup>	<60 <sup>b</sup>	<20 <sup>b</sup>	NA	<50 <sup>b</sup>	<50 <sup>b</sup>	<1 <20
RRPH (Approx.)	12-30	120-300	2,000 <sup>a</sup>	<120 <300	<160	<120	<240	<300	<120	NA	<2,000	NA	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1 <0.3	<0.2	<0.15	<0.2	<0.3	<0.1				
Benzene	0.002-0.006	0.02-0.06	0.5	<0.02 <0.06	<0.04	<0.03	<0.04	<0.06	<0.02	<1 <sup>c</sup>	<1	5.0N	<0.02 <0.2
Toluene	0.002-0.006	0.02-0.06		<0.02 <0.06	<0.04	<0.03	<0.04	<0.06	<0.02	<1 <sup>c</sup>	<1	1N	<0.02 <0.2
Ethylbenzene	0.002-0.006	0.02-0.06		<0.02 <0.06	<0.04	<0.03	<0.04	<0.06	<0.02	<1 <sup>c</sup>	<1	1N	<0.02 <0.2
Xylenes (Total)	0.004-0.012	0.04-0.12		<0.04 <0.12	<0.06	<0.06	<0.06	<0.12	<0.04	<2 <sup>c</sup>	<2	5.0N	<0.04 <0.4
HVOC 8010	0.002-0.006	0.02-0.06		<0.02 <0.06	<0.04	<0.03	<0.04	<0.06	<0.02	NA	<1.5 <10J	<1.5	<0.02
VOC 8260													
Methylene Chloride	0.020	0.025-0.160	90	<0.025 <0.160	<0.040	<0.030	<0.160	<0.1	<0.025	13J	<1	1.7	<0.020
SVOC 8270													
di-n-Butylphthalate	0.200	0.250-4.23	8,000	1.61U-20.4JB	1.61U	2.19U	NA	NA	20.4JB	NA	<10	NA	0.87B
Pesticides													
Dieldrin	0.001-1	0.01-10	0.04	<0.01 <0.07R	0.07R	<0.01	<0.01	<0.01	<0.01	NA	<0.2 <10J	NA	<0.01 <0.5

CT&E Data.

F&B Data.

Not analyzed.

The analyte was detected in the associated blank.

The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

Result has been rejected.

Compound is not present above the concentration listed.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.



TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Background (BKGD)		Matrix: Soil/Sediment Units: mg/kg											
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples				Field Blanks			Lab Blanks	
					S01	S02	S03	S04	SD01	AB01	EB01		TB01
Laboratory Sample ID Numbers					1382/1554 4477-1 4478-2	1555/1556 4481-4	1574 4481-5	1576 4481-6	1381/1557 4477-2 4478-3	4512-3	1558 1581 4478-5	1552 4478-1	#3&4-9483 #6-9593 #6-9283 4481\4478\4477
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	mg/kg
PCBs													
Aroclor 1260	0.01	0.1	10	<0.02-20JIN	20JIN	<0.1	<0.2	<0.3	<0.1	NA	<2J	NA	<0.1
TOC				25,700-77,900	77,900	NA	NA	NA	25,700	NA	NA	NA	NA

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

**TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Background (BKGD)		Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES										Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples					Field Blank					
					S01	S02	S03	S04	SD01		EB01				
Laboratory Sample ID Numbers					4477-1	4481-4	4481-5	4481-6	4477-2		4476-5			4477 4476 4481	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg										µg/L	
Aluminum	0.35	2		4,700-17,000	4,700	17,000	5,000	10,500	6,300		<100			<100	
Antimony	N/A	61-110		<61-<110	<95J	<61	<110	<69	<63J		<100			<100	
Arsenic	0.11	6.3-69		<6.3-<69	<9.5	<61	<11	<69	<6.3		<100			<100	
Barium	0.024	1		590-2,000	590	1,100	940	2,000	800		<50			<50	
Beryllium	N/A	3.2-35		<3.2-<35	<4.8	<31	<5.4	<35	<3.2		<50			<50	
Cadmium	0.33	31-54		<31-<54	<48	<31	<54	<35	<32		<50			<50	
Calcium	0.69	4		2,700-240,000	240,000	2,700	26,000	8,900	8,500		210			<200	
Chromium	0.066	1		9.3-33	9.3	33	13	24	11		<50			<50	
Cobalt	N/A	1-11		<6.3-17	11	13	<11	17	<6.3		<100			<100	
Copper	0.045	1		12-71	16	36	71	50	12		<50			<50	
Iron	0.50	2		5,400-39,000	25,000	28,000	11,000	39,000	5,400		110			<100	
Lead	0.13	2-69		<9.5-7.0	<9.5	<61	<11	<69	7.0		<100			<100	
Magnesium	0.96	4		1,000-34,000	34,000	5,100	2,400	2,000	1,000		<200			<200	
Manganese	0.025	1		15J-1,000J	1,000J	270	780	910	15J		<50			<50	
Molybdenum	N/A	3.1-35		<3.1-<35	<4.8	<3.1	<5.4	<35	<3.2		<50			<50	
Nickel	0.11	1		13-80	33	60	80	34	13		<50			<50	
Potassium	23	100-540		<540-2,600	2,100	2,600	<540	570	640		<5,000			<5,000	

☐ CT&E Data.  
☐ N/A  
☐ Not available.  
☐ Result is an estimate.

**TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Background (BKGD)			Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples					Field Blank			Lab Blanks	
					S01	S02	S03	S04	SD01		EB01			
Laboratory Sample ID Numbers					4477-1	4481-4	4481-5	4481-6	4477-2			4476-5	4477 4476 4481	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg			µg/L	µg/L	
Selenium	1.2	61-110		<61-<110	<95	<61	<110	<69	<63			<100	<100	
Silver	0.53	31-54		<31-<54	<48J	<31	<54	<35	<32J			<50	<50	
Sodium	0.55	5		58-120	89	100	120	110	58			360	<250	
Thallium	0.011	0.31-0.52		<0.31-<0.52	<0.51	<0.31	<0.52	<0.35	<0.31			<5	<5	
Vanadium	0.036	1		19-58	19	46	32	58	21			<50	<50	
Zinc	0.16	1		40J-250	130J	130	250	67	40J			<50	<50	

☐ CT&E Data.  
J Result is an estimate.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Background (BKGD)		Matrix: Sediment Units: mg/kg		Environmental Samples						Field Blank	Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	5SD01	5SD02	5SD03-2	5SD04	5SD05	3EB02	Lab Blanks
Laboratory Sample ID Numbers					2592-1	2592-2	2592-3	2592-4	2592-5	4742-2	2592
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	mg/kg
PCBs	0.02	0.02-0.04	10	<0.02-20.0	<0.02	<0.03	<0.04	<0.02	<0.02	<1	<0.02

□ CT&E Data.

■ F&B Data.

NA Not available.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Background (BKGD)		Matrix: Surface Water Units: µg/L		Environmental Samples			Field Blanks			Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	SW01	SW02	AB01	EB01	TB01	
Laboratory Sample ID Numbers					1562/1564 4477-5 4476-6	1566/1568 4477-6 4476-7	4512-3	1558/1561 4476-5	1552	#1&2-9493 #1&2-9793 #6-9393 4512 4476 4477
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	<100	<1,000		<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<2,000
GRPH	<5	<50		<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	NA	<50 <sup>b</sup>	<50 <sup>b</sup>	<50
RRPH (Approx.)	<200	<2,000		<2,000	<2,000	<2,000	NA	<2,000	NA	<4,000
BTEx (8020/8020 Mod.)										
Benzene	0.1-1	1	5	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1
Toluene	0.1-1	1	1,000	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1
Ethylbenzene	0.1-1	1	700	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1
Xylenes (Total)	0.2-2	2	10,000	<2 <sup>c</sup>	<2 <sup>c</sup>	<2 <sup>c</sup>	<2 <sup>c</sup>	<2 <sup>c</sup>	<2 <sup>c</sup>	<2
HVOC 8010	0.1-1	1		<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	NA	<1 <sup>c</sup> <10 <sup>c</sup>	<1 <sup>c</sup>	<1-<10 <sup>c</sup>
VOC 8260										
Methylene Chloride	1	1	5	<1	<1	<1	13J	<1	1.7	<1
SVOC 8270	10	10		<10	<10	<10	NA	<10	NA	<10

□

CT&amp;E Data.

■

F&amp;B Data.

NA

Not analyzed.

J

Result is an estimate.

N

The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

a

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c

BTEx determined by 8260 method analysis.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Background (BKGD)		Matrix: Surface Water Units: µg/L		Environmental Samples			Field Blanks			Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	SW01	SW02	AB01	EB01	TB01	
Laboratory Sample ID Numbers					1562/1564 4477-5 4476-6	1566/1568 4477-6 4476-7	4512-3	1558/1561 4476-5	1552	#1&2-9493 #1&2-9793 #6-9393 4512 4476 4477
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
Pesticides	0.02-1	0.2-10		<0.2J-<10J	<0.2J-<10J	<0.2-<10	NA	<0.2J-<10J	NA	NA
PCBs	0.2	2	0.5	<2J	<2J	<2J	NA	<2J	NA	NA
TOC	5,000	5,000		<5,000-15,600	<5,000	15,600	NA	NA	NA	<5,000
TSS	100	200		2,500-3,000	2,500	3,000	NA	NA	NA	<200
TDS	10,000	10,000		203,000-245,000	245,000	203,000	NA	NA	NA	<10,000

☐ CT&E Data.  
☒ F&B Data.  
☐ Not analyzed.  
 J Result is an estimate.

**TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Background (BKGD)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples					Field Blank		Lab Blanks
					SW01	SW02						
Laboratory Sample ID Numbers					4477-5	4477-6					4476-5	4477 4476
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L	µg/L
Aluminum	17.4	100		<100 (<100)	<100 (<100J)	<100 (<100)					<100	<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100
Barium	1.2	50	2,000	79-92 (73-89)	92 (89)	79 (73)					<50	<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Calcium	34.5	200		28,000-41,000 (28,000-41,000)	41,000 (41,000)	28,000 (28,000)					210	<200
Chromium	3.3	50	100	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)					<100	<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Iron	25.0	100		<100-190 (<100)	<100 (<100)	190 (<100)					110	<100
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100

☐ CT&E Data.  
☐ N/A Not analyzed.  
☐ J Result is an estimate.

**TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Background (BKGD)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples					Field Blank		Lab Blanks
					SW01	SW02						
Laboratory Sample ID Numbers					4477-5	4477-6					4476-5	4477 4476
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L	µg/L
Magnesium	47.8	200		4,500-9,000 (4,500-8,800)	9,000 (8,800)	4,500 (4,500)					<200	<200
Manganese	1.2	50		<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)					<5,000	<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Sodium	27.7	250		4,000-5,600 (3,900-6,000)	4,000 (3,900)	5,600 (6,000)					360	<250
Thallium	0.571	5	2	<5 (<5)	<5 (<5)	<5 (<5)					<5	<5
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)					<50	<50
Zinc	8.2	50		<50-260 (<50)	260 (<50)	<50 (<50)					<50	<50

☐ CT&E Data.  
N/A Not analyzed.



TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)										Matrix: Soil Units: mg/kg		
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks		Lab Blanks
					S01	S02	S03-4	S04-4	S05-5	AB01	TB02	
Laboratory Sample ID Numbers					1594	1596	1598	1600	1602 4514-11	4512-3	1514 4512-2	#6-9393 #182-9493 4512 4514
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L
DRPH	5-8	50-80	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<80 <sup>b</sup>	<60 <sup>b</sup>	<50 <sup>b</sup>	<70 <sup>b</sup>	NA	NA	NA	<2,000
GRPH	0.2-0.3	2-3	100	<2 <sup>b</sup> <8 <sup>b</sup>	<3 <sup>b</sup>	<3 <sup>b</sup>	<2 <sup>b</sup>	<3 <sup>b</sup>	5 <sup>b</sup>	NA	<50 <sup>b</sup>	<50
RRPH (Approx.)	10-20	100-200	2,000 <sup>a</sup>	<120 <300	<200	<200	<100	<200	NA	NA	NA	<4,000
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1 <0.3	<0.15	<0.15	<0.1	<0.1	3.1J			
Benzene	0.002-0.003	0.02-0.03	0.5	<0.02 <0.06	<0.03	<0.03	<0.02	<0.02	<0.02	NA	<1	<0.02 <0.2
Toluene	0.002-0.003	0.02-0.03		<0.02 <0.06	<0.03	<0.03	<0.02	<0.02	<0.02	NA	<1	<0.02 <0.2
Ethylbenzene	0.002-0.003	0.02-0.03		<0.02 <0.06	<0.03	<0.03	<0.02	<0.02	0.8	NA	<1	<0.02 <0.2
Xylenes (Total)	0.004-0.008	0.04-0.08		<0.04 <0.12	<0.06	<0.06	<0.04	<0.04	2.5J	NA	<2	<0.04 <0.4
HVOC 8010				<0.02J <0.06J	<0.03J	<0.03J	<0.02J	<0.02J	0.1J	NA	<1J	<0.02J
Trichloroethene	0.002-0.003	0.02-0.03										
VOC 8260												
cis-1,2-Dichloroethene	0.020	0.025		<0.025 <0.160	NA	NA	NA	NA	0.048	<1	<1	<0.020
p-Isopropyltoluene	0.020	0.025		<0.025 <0.160	NA	NA	NA	NA	0.113	<1	<1	<0.020
Trichloroethene	0.020	0.025		<0.025 <0.160	NA	NA	NA	NA	0.390	<1	<1	<0.020
1,3,5-Trimethylbenzene	0.020	0.025		<0.025 <0.160	NA	NA	NA	NA	0.285	<1	<1	<0.020
SVOC 8270	0.200	7.98		1.61U-20.4JB	NA	NA	NA	NA	<7.98	NA	NA	<10
												0.878

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

B The analyte was detected in the associated blank.

J Result is an estimate.

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.



TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)												Matrix: Soil Units: mg/kg	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blanks					Lab Blanks	
					S08	2S11	AB01	2EB04	EB02	2TB04	TB02		
Laboratory Sample ID Numbers					1608	1923 4728-8	4512-3	1924 4727-10	1510/1542 4512-1	4727-9	1514 4512-2	#6-9393 #1&2-9493 4512	#3&4-9493 #6-9593 4514
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	mg/kg
DRPH	14-15	140-150	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<150 <sup>b</sup>	<140 <sup>b</sup>	NA	<1,000 <sup>b</sup>	<1,000J <sup>b</sup>	NA	NA	<2,000	<50
GRPH	0.400-0.8	0.400-8	100	<2J <sup>b</sup> <6J <sup>b</sup>	<8J <sup>b</sup>	<1.80	NA	<20	<50J <sup>b</sup>	NA	<50J <sup>b</sup>	<50	<2J <20
RRPH (Approx.)	30-40	300-400	2,000 <sup>a</sup>	<120 <300	<400	<300	NA	<4,000	<2,000	NA	NA	<4,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1 <0.3	<0.30								
Benzene	0.006	0.06	0.5	<0.02 <0.06	<0.06	NA	NA	NA	<1 <sup>c</sup>	NA	<1	<1	<0.02 <0.2
Toluene	0.006	0.06		<0.02 <0.06	<0.06	NA	NA	NA	<1 <sup>c</sup>	NA	<1	<1	<0.02 <0.2
Ethylbenzene	0.006	0.06		<0.02 <0.06	<0.06	NA	NA	NA	<1 <sup>c</sup>	NA	<1	<1	<0.02 <0.2
Xylenes (Total)	0.012	0.12		<0.04 <0.12	<0.12	NA	NA	NA	<2 <sup>c</sup>	NA	<2	<2	<0.04 <0.4
HVOC 8010	0.006	0.06		<0.02J <0.06J	<0.06J	NA	NA	NA	<1J	NA	<1J	<1	<0.02J
VOC 8260													
Toluene	0.020	0.090		<0.025 <0.160	NA	0.300	<1	<1J	<1	<1J	<1	<1	<0.020
1,2,4-Trimethylbenzene	0.020	0.090		<0.025 <0.160	NA	0.305	<1	<1J	<1	<1J	<1	<1	<0.020
Xylenes (Total)	0.040	0.180		<0.050 <0.320	NA	0.522	<2	<2J	<2	<2J	2	<2	<0.040

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

d The samples were collected prior to interim remedial action (clean up) field activities; site conditions have since changed.

TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne				Matrix: Soil								
Site: Landfill and Waste Accumulation Area (LF01)				Units: mg/kg								
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blanks				Lab Blanks	
					S08	2S11	AB01	2EB04	EB02	2TB04	TB02	
Laboratory Sample ID Numbers					1608	1923 4728-8	4512-3	1924 4727-10	1510/1542 4512-1	4727-9	1514 4512-2	#6-9393 #1&2-9493 #6-9593 4514
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	µg/L	mg/kg
SVOC 8270												
di-n-Butylphthalate	0.200	11.8		1.61U-20.4JB	NA	30.3B	NA	NA	<11	NA	NA	0.878
PCBs	0.4	0.1	10	<0.02-20JN	<4	NA	NA	<2	<2J	NA	NA	<0.1

☐ CT&E Data.  
☒ F&B Data.  
☒ NA  
 B Not analyzed.  
 J The analyte was detected in the associated blank.  
 N Result is an estimate.  
 U The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".  
 Compound is not present above the concentration listed.



TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)										Matrix: Soil Units: mg/kg			
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks		
					4S35-2	4S40-5	4S44-4	LF01 AB01	3EB02	4TB01	1850 4742 4762	1850 mg/kg	
Laboratory Sample ID Numbers					1850-2	1850-3	1850-4	4762-1	4742-2	1850-1			
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg	
DRPH	4.00	4.00	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	441	2,512	1,780	NA	NA	NA	NA	<4.00	
GRPH	0.400	0.400	100	<2J <sup>b</sup> <6J <sup>b</sup>	4.88	50.9	8.64	NA	NA	NA	NA	<1.00	
RRPH (Approx.)	4.00	4.00	2,000 <sup>a</sup>	<120 <300	2,000	5,030	4,240	NA	NA	NA	NA	<100	
VOC 8260													
n-Butylbenzene	0.020	0.100		<0.025 <0.160	<0.100	0.636	<0.100	<1	NA	<1	<1	<0.050	
sec-Butylbenzene	0.020	0.100		<0.025 <0.160	<0.100	0.168	<0.100	<1	NA	<1	<1	<0.050	
Carbon Tetrachloride	0.020	0.100		<0.025 <0.160	0.110	17.3	<0.100	<1	NA	<1	<1	<0.050	
Chloroform	0.020	0.100		<0.025 <0.160	<0.100	0.796	<0.100	<1	NA	<1	<1	<0.050	
Ethylbenzene	0.020	0.100		<0.025 <0.160	<0.100	2.19	<0.100	<1	NA	<1	<1	<0.050	
Isopropylbenzene	0.020	0.100		<0.025 <0.160	<0.100	0.242	<0.100	<1	NA	<1	<1	<0.050	
p-Isopropyltoluene	0.020	0.100		<0.025 <0.160	<0.100	0.178	<0.100	<1	NA	<1	<1	<0.050	
Naphthalene	0.020	0.100		<0.025 <0.160	<0.100	0.185	0.125	<1	NA	<1	<1	<0.050	
n-Propylbenzene	0.020	0.100		<0.025 <0.160	<0.100	0.758	<0.100	<1	NA	<1	<1	<0.050	
Toluene	0.020	0.100		<0.025 <0.160	<0.100	3.11	8.45	<1	NA	<1	<1	<0.050	
Trichloroethene	0.020	0.100		<0.025 <0.160	<0.100	15.3	0.175	<1	NA	<1	<1	<0.050	

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

a The action levels for DRPH and RRPB are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne		Matrix: Soil									
Site: Landfill and Waste Accumulation Area (LF01)		Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
					4S35-2	4S40-5	4S44-4	LF01 AB01	3EB02	4TB01	
Laboratory Sample ID Numbers					1850-2	1850-3	1850-4	4762-1	4742-2	1850-1	1850 4742 4762
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	mg/kg
1,2,4-Trimethylbenzene	0.020	0.100		<0.025-<0.160	<0.100	6.89	0.486	<1	NA	<1	<0.050
1,3,5-Trimethylbenzene	0.020	0.100		<0.025-<0.160	<0.100	2.04	<0.100	<1	NA	<1	<0.050
Xylenes (Total)	0.040	0.200		<0.050-<0.320	<0.200	9.83	0.824	<2	NA	<2	<0.100
SVOC 8270											
2-Methylnaphthalene	0.20	0.353-5.26		<0.250-<4.23	<0.353	8.59	0.229J	NA	NA	NA	<0.100
4-Methylphenol	0.20	0.353-5.26		<0.250-<4.23	<0.353	<5.26	2.16	NA	NA	NA	<0.100
Naphthalene	0.20	0.353-5.26		<0.250-<4.23	<0.353	4.26J	<0.354	NA	NA	NA	<0.100
Phenol	0.20	0.353-5.26		<0.250-<4.23	<0.353	<5.26	0.920	NA	NA	NA	<0.100
PCBs											
Aroclor 1260	0.020	0.08	10	<0.02-20JN	<0.08	0.149	0.100	NA	<1	NA	<1

☐ CT&E Data.  
☒ F&B Data.  
☒ Not analyzed.  
 NA Result is an estimate.  
 J The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".  
 N

TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)													Matrix: Soil Units: mg/kg	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks	
					5S55	5S58	5S65	5S68	5S70	LF01 AB01	3EB02	5TB02		
Laboratory Sample ID Numbers					2610-1	2610-2	2610-3	2610-4	2610-5	4762-1	4742-2	2610-10	2610 4742 4762	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	mg/kg	
DRPH	4.00	4.00	500 <sup>3</sup>	<60 <sup>3</sup> <150 <sup>3</sup>	NA	NA	NA	18,600	NA	NA	NA	NA	<4.00	
GRPH	0.400	0.400	100	<25 <sup>3</sup> <50 <sup>3</sup>	NA	NA	NA	17.2	NA	NA	NA	NA	<1.00	
RRPH (Approx.)	4.00	4.00	2,000 <sup>3</sup>	<120 <300	NA	NA	NA	43,100	NA	NA	NA	NA	<100	
VOC 8260														
Methylene Chloride	0.020	0.050		<0.025-<0.160	NA	NA	NA	0.066	NA	<1	NA	<1	<0.050	
Naphthalene	0.020	0.050		<0.025-<0.160	NA	NA	NA	0.147	NA	<1	NA	<1	<0.050	
1,2,4-Trimethylbenzene	0.020	0.050		<0.025-<0.160	NA	NA	NA	0.166	NA	<1	NA	<1	<0.050	
1,3,5-Trimethylbenzene	0.020	0.050		<0.025-<0.160	NA	NA	NA	0.154	NA	<1	NA	<1	<0.050	
PCBs														
Aroclor 1260	0.020	0.020	10	<0.02-200UN	29.7	11.4	0.999	0.798	7.91	NA	<1	NA	<1	<0.020

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.



TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne		Matrix: Soil		Units: mg/kg			
Site: Landfill and Waste Accumulation Area (LF01)							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blank
					5S71	5S72	5S73
Laboratory Sample ID Numbers					2610-6	2610-7	2610-8
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
PCBs							
Aroclor 1260	0.020	0.020	10	<0.02-20JN	59.7	153	0.472
							<1
							<1
							4742
							μg/L
							mg/kg
							mg/kg
							0.020

☐ CT&E Data.  
☒ F&B Data.  
 Result is an estimate.  
 The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne																
Site: Landfill and Waste Accumulation Area (LF01)																
Matrix: Sediment																
Units: mg/kg																
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bgkd. Levels	Environmental Samples						Field Blanks			Lab Blanks		
					SD01	SD02	SD03 & SD08 (Replicates)	SD04	SD05	SD06	AB01	EB02	TB02			
Laboratory Sample ID Numbers					1578	1580	1582 4514-2	1592 4514-7	1584	1588 4514-6	1588	4512-3	1510/1542 4513-1 4512-1	1514 4512-2	#6-9393 #182-9493 4512	#6-9393 #384-9493 4512
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	mg/kg
DRPH	5-56	50-560	500 <sup>a</sup>	<80 <sup>a</sup> <150 <sup>b</sup>	<140 <sup>b</sup>	<80 <sup>b</sup>	<100 <sup>b</sup>	<80 <sup>b</sup>	<560 <sup>b</sup>	<360 <sup>b</sup>	1,200 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	NA	<50
GRPH	0.2-4.0	2-40	100	<2J <sup>b</sup> <8J <sup>b</sup>	<8J <sup>b</sup>	<2J <sup>b</sup>	<4J <sup>b</sup>	<3J <sup>b</sup>	<40J <sup>b</sup>	<130J <sup>b</sup>	<5J <sup>b</sup>	NA	<50J <sup>b</sup>	<50J <sup>b</sup>	<50	<2J-<20
RRPH (Approx.)	12-110	120-1,100	2,000 <sup>a</sup>	<120 <300	<280	<120	<200	<200	<1,100	<710	5,000	NA	<2,000	NA	NA	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.3	<0.3	<0.1	<0.2	<0.15	<1.0	<12.4	<0.25					
Benzene	0.002-0.02	0.02-0.2	0.5	<0.02 <0.06	<0.06	<0.02	<0.04	<0.03	<0.2	<0.2	<0.05	<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2
Toluene	0.002-0.02	0.02-0.2		<0.02 <0.06	<0.06	<0.02	<0.04	<0.03	<0.2	<0.2	<0.05	<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2
Ethylbenzene	0.002-0.5	0.02-5		<0.02 <0.06	<0.06	<0.02	<0.04	<0.03	<0.2	<5	<0.05	<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2
Xylenes (Total)	0.004-0.7	0.04-7		<0.04 <0.12	<0.12	<0.04	<0.08	<0.06	<0.4	<7	<0.1	<2 <sup>c</sup>	<2	<2	<2	<0.04-<0.4
HVOC 8010	0.002-0.02	0.02-0.2		<0.02J-<0.06J	<0.06J	<0.02J	<0.04J	<0.03J	<0.2J	<0.2J	<0.05J	NA	<1J	<1J	<5-<10	<0.02J
VOC 8260				<0.025-<0.160	NA	NA	<0.050	<0.040	NA	0.241	NA	<1	<1	<1	<1	<0.020
Toluene	0.020	0.040-0.160														
SVOC 8270																
di-n-Butyl-phthalate	0.200	6.23-29	8,000	1.61U-20.4JB	NA	NA	7.53B	<6.23	NA	<29.0	NA	NA	<11	NA	<10	0.878
PCBs	0.01	0.1	10	<0.02-20JIN	<0.2	<0.1	<0.2	<0.1	<1.1	<0.7	<0.3	NA	<2J	NA	NA	<0.1

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

B The analyte was detected in the associated blank.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

U Compound is not present above the concentration listed.

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)				Matrix: Sediment Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Sample			Field Blanks				Lab Blanks	
					SD07	3SD23		AB01	EB02	3EB02	TB02		
Laboratory Sample ID Numbers					1590	4608-6		4512-3	1510/1542	4742-2	1514	#6-9393 #1&2-9493 4512	#6-9593 #3&4-9493
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg		μg/L	μg/L	μg/L	μg/L	μg/L	μg/L
DRPH	50	500	500 <sup>a</sup>	<60 <sup>b</sup> , <150 <sup>b</sup>	<500 <sup>b</sup>	NA	NA	<1,000J <sup>b</sup>	NA	NA	NA	NA	<50
GRPH	2	20	100	<2J <sup>b</sup> , <6J <sup>b</sup>	<20J <sup>b</sup>	NA	NA	<50J <sup>b</sup>	NA	NA	<50J <sup>b</sup>	<50	<1-<20
RRPH (Approx.)	50	500	2,000 <sup>a</sup>	<120-<300	4,700	NA	NA	<2,000	NA	NA	NA	NA	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.3	<1.0								
Benzene	0.02	0.2	0.5	<0.2-<0.6	<0.2	NA	<1 <sup>c</sup>	<1	NA	NA	<1	<1	<0.02-<0.2
Toluene	0.02	0.2		<0.2-<0.6	<0.2	NA	<1 <sup>c</sup>	<1	NA	NA	<1	<1	<0.02-<0.2
Ethylbenzene	0.02	0.2		<0.2-<0.6	<0.2	NA	<1 <sup>c</sup>	<1	NA	NA	<1	<1	<0.02-<0.2
Xylenes (Total)	0.04	0.4		<0.04-<0.12	<0.4	NA	<2 <sup>c</sup>	<2	NA	NA	<2	<2	<0.04-<0.4
HVOC 8010	0.02	0.2		<0.2J-<0.6J	<0.2J	NA	NA	<1J	NA	NA	<1J	<1	<0.02
PCBs													
Aroclor 1254	0.020-0.1	0.020-1	10	<0.02-<0.3	<1J	0.235	NA	<2J	<1	NA	NA	NA	<0.1

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)		Matrix: Soil/Sediment Units: mg/kg		Environmental Samples				Field Blank	Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	2SD09 & 2SD13 (Replicates)	2SD10	2SD11	2SD12	AB01
Laboratory Sample ID Numbers					4728-1	4728-4	1920 4728-5	4728-6	#6-91393 4727 4512
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/L
DRPH	14-26	140-260	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	NA	500 J <sup>b</sup>	<260 <sup>b</sup>	NA	<1,000
GRPH	0.400	0.400-6	10	<2 J <sup>b</sup> <6 J <sup>b</sup>	0.665	<2	<6	<5	<50
RRPH (Approx.)	30-53	300-530	2,000 <sup>a</sup>	<120 <300	NA	2,300	<530	NA	<4,000
VOC 8260									
Benzene	0.020	0.035-0.300	0.5	<0.025-<0.160	<0.035J	0.104	<0.300	0.256	<1
Ethylbenzene	0.020	0.035-0.300		<0.025-<0.160	0.060J	0.225	0.400	0.690	<1
n-Propylbenzene	0.020	0.035-0.300		<0.025-<0.160	<0.035J	<0.100	<0.300	0.315	<1
Toluene	0.020	0.035-0.300		<0.025-<0.160	0.179J	0.877	1.50	2.52	<1
1,2,4- Trimethylbenzene	0.020	0.035-0.300		<0.025-<0.160	0.133J	0.533	0.949	1.73	<1
1,3,5- Trimethylbenzene	0.020	0.035-0.300		<0.025-<0.160	0.043J	0.189	0.321	0.600	<1
Xylenes (Total)	0.040	0.070-0.600		<0.05-<0.320	0.393J	1.484	2.651	4.59	<1
SVOC 8270									
di-n-Butylphthalate	0.200	3.03-20.9	8,000	1.61U-20.4UB	7.02U	NA	26.3B	55.8B	27.6B
									NA
									2.310

☐ CT&E Data.

☒ F&B Data.

☒ Not analyzed.

☒ The analyte was detected in the associated blank.

☒ Result is an estimate.

☒ Compound is not present above the concentration listed.

☒ The action levels for DRPH and RRRPH are based on conversations with ADEC; final action levels have not yet been determined.

☒ DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)			Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES						
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						
					SD03 & SD08 (Replicates)	SD05	S05	S06 & S09 (Replicates)		Field Blank	Lab Blanks
Laboratory Sample ID Numbers					4514-2	4514-7	4514-6	4514-11	4514-12	4514-13	
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	
Aluminum	0.35	2		4,700-17,000	4,900J	2,800J	5,200	6,000	6,600	8,200	4511-1
Antimony	N/A	2-75		<61-<110	<47R	<75R	<88	<59R	<68	<61	<100
Arsenic	0.11	8.8-75		<6.3-<69	<47	<75	<8.8	<59	<68	<61	<100
Barium	0.024	1		590-2,000	630	540	430	230	500	400	<50
Beryllium	N/A	1		<3.2-<31	<2.4	<3.8	<4.4	<3.0	<3.4	<3.1	<50
Cadmium	0.33	1		<31-<54	<24	<38	<44	<30	<34	<31	<50
Calcium	0.69	4		2,700-240,000	19,000	31,000	11,000	41,000	6,300	5,500	<200
Chromium	0.066	1		9.3-33	9.0	5.4	8.9	9.8	10	13	<50
Cobalt	N/A	1-8.8		<6.3-17	5.6J	<7.5J	<8.8	<5.9	<6.8	<6.1	<100
Copper	0.045	1		12-71	9.1	5.7	13	8.5	26	20	<50
Iron	0.50	2		5,400-39,000	18,000	14,000	12,000	8,100	9,300	15,000	<100
Lead	0.13	8.8-75		<9.5-7.0	<47	<75	<8.8	<59	<68	<61	<100
Magnesium	0.96	4		1,000-34,000	4,200	5,300	2,100	11,000	1,300	1,700	<200
Manganese	0.025	1		15J-1,000J	750J	260J	160	150	100	88	<50
Molybdenum	N/A	2.4-4.4		<3.1-<35	<2.4	<3.8	<4.4	<3.0	<3.4	<3.1	<50
Nickel	0.11	1		13-80	15	11	17	12	12	15	<50
Potassium	23	100-440		<540-2,600	540J	<370J	<440	1,370	680	930	<5,000

☐ CT&E Data.  
☐ N/A Not available.  
☐ J Result is an estimate.  
☐ R Result has been rejected.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)				Matrix: Soil/Sediment Units: mg/kg		METALS ANALYSES							
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blank		Lab Blanks	
					SD03 & SD08 (Replicates)	SD05	S05	S06 & S09 (Replicates)			EB02		
Laboratory Sample ID Numbers					4514-2	4514-7	4514-6	4514-11	4514-12	4514-13			4514 4511
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg			µg/L
Selenium	1.2	47-88		<61-<110	<47	<75	<88	<59	<68	<61		<100	<100
Silver	0.53	24-44		<31-<54	<24R	<38R	<44	<30R	<34	<31		<50J	<50
Sodium	0.55	5		58-120	110	97	290	150	130	150		340	<250
Thallium	0.011	0.23-0.44		<0.31-<0.52	<0.23J	<0.39J	<0.44	<0.29J	<0.31	<0.36		<5	<5
Vanadium	0.036	1		19-58	18	14	18	17	25	28		<50	<50
Zinc	0.16	1		40J-250	49	36	57	73	70	45		<50	<50

☐ CT&E Data.  
☐ J Result is an estimate.  
☐ R Result has been rejected.

TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)					Matrix: Sediment Units: mg/kg						
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blank	Lab Blanks
					5SD01	5SD02	5SD03-1	5SD04	5SD05		
Laboratory Sample ID Numbers					2592-8	2592-9	2592-10	2592-11	2592-12	4742-2	2592
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	mg/kg
PCBs	0.02	0.02-0.05	10	<0.02-20 µg/g	<0.02	<0.03	<0.05	<0.03	<0.04	<1	<0.02

☐ CT&E Data.  
☒ F&B Data.  
 Result is an estimate.  
 The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)		Matrix: Sediment Units: mg/kg		Environmental Samples				Field Blank	Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	5SD06	5SD07	5SD08	5SD09	
Laboratory Sample ID Numbers					2592-13	2592-14	2592-15	2592-16	2592
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
PCBs									
Aroclor 1260	0.020	0.02-0.04	10	<0.02-200 N	<0.04	<0.02	0.309	7.42	<1
									<0.02

☐ CT&E Data.  
☒ F&B Data.  
 Result is an estimate.  
 The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".





TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)					Matrix: Surface Water Units: µg/L					Environmental Samples					Field Blanks			Lab Blanks
Parameter/s	Defect. Limits	Quant. Limits	Action Levels	Bkqd. Levels	SW01	SW02	SW03	SW04	SW05	SW06 & SW07 (Duplicates)		AB01	EB02	TB02				
Laboratory Sample ID Numbers					1516/1543	1518/1544 4514-1 4512-4	1522/1545	1524/1546 4512-8 4514-8	1534/1549 4512-5 4514-14	1536/1550 4512-9 4514-5	1540/1551 4512-10 4514-16	4512-3	1510/1542 4512-1	1514 4512-2	#6-9393 #182-9493 4512 4514			
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			
DRPH	100	1,000		<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	NA			
GRPH	5	50		<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	NA	<50 <sup>b</sup>	<50 <sup>b</sup>	<50			
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000	<2,000	<2,000	<2,000	<2,000	NA	<2,000	NA	NA			
BTEX (8020/8020 Mod.)																		
Benzene	0.1	1	5	<1 <sup>c</sup>	5J	<1	5J	<1	<1	<1	<1	<1 <sup>c</sup>	<1	<1	<1			
Toluene	0.1	1	1,000	<1 <sup>c</sup>	<1	<1	2	<1	<1	1	<1	<1 <sup>c</sup>	<1	<1	<1			
Ethylbenzene	0.1	1	700	<1 <sup>c</sup>	2	<1	2	<1	<1	1	1	<1 <sup>c</sup>	<1	<1	<1			
Xylenes (Total)	0.2	2	10,000	<2 <sup>c</sup>	5J	<2	5J	<2	<2	4J	4J	<2 <sup>c</sup>	<2	<2	<2			
HVOC 8010																		
Carbon Tetrachloride	0.1-0.2	1-2	5	<0.02J-<0.06J	<1J	<1J	<1J	<1J	<1J	1J	2J	NA	<1J	<1J	NA			
Tetrachloroethene	0.1-0.2	1-2	5	<0.02J-<0.06J	<1J	<1J	4J	<1J	<1J	<1J	<1J	NA	<1J	<1J	NA			
Trichloroethene	0.1-0.2	1-2	5	<0.02J-<0.06J	<1J	<1J	<1J	<1J	<1J	1J	2J	NA	<1J	<1J	NA			
VOC 8260																		
Carbon Tetrachloride	1	1	5	<1	NA	<1	NA	<1	NA	48	1.4	<1	<1	<1	<1			
Chloroform	1	1		<1	NA	<1	NA	<1	NA	4.8	2.4	<1J	<1	<1	<1			

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne		Matrix: Surface Water										Lab	
Site: Landfill and Waste Accumulation Area (LF01)		Units: µg/L										Blanks	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blanks		
					SW01	SW02	SW03	SW04	SW05	SW06 & SW07 (Duplicates)	AB01	EB02	TB02
Laboratory Sample ID Numbers					1516/1543	1518/1544 4514-1 4512-4	1522/1545	1524/1546 4512-5 4514-8	1534/1549 4512-8 4514-14	1536/1550 4512-9 4514-5	4512-3	1510/1542 4512-1	1514 4512-2 4514
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
cis-1,2-Dichloroethene	1	1	70	<1	NA	<1	NA	<1	NA	1.2	<1J	<1	<1
Trichloroethene	1	1	5	<1	NA	<1	NA	<1	NA	6.8	<1	<1	<1
SVOC 8270	10	10-31		<10	NA	<11	NA	<31	<10	<11	NA	<11	<10
Pesticides	0.02-1	0.2-10		<0.2J - <10J	NA	NA	NA	<0.2J - <10	NA	NA	NA	<0.2J - <10	NA
PCBs	0.2	2	0.5	<2J	<2J	<2J	<2J	<2J	<2J	<2J	NA	<2J	NA
TOC	5,000	5,000		<5,000-15,600	NA	10,200	NA	24,500	52,900	32,100	NA	NA	<5,000
TSS	100	200		2,500-3,000	NA	2,500	NA	56,000	13,000	35,000	NA	NA	<200
TDS	10,000	10,000		203,000-245,000	NA	236,000	NA	328,000	688,000	258,000	NA	NA	<10,000

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne				Matrix: Surface Water								
Site: Landfill and Waste Accumulation Area (LF01)				Units: µg/L								
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks		Lab Blanks		
					2SW08	2SW09 & 2SW10 (Duplicates)		AB01	2EB04		2TB04	
Laboratory Sample ID Numbers					1915 4727-4 4729-1	1917 4727-7 4729-2	1918 4727-8 4729-3		4512-3	1924 4727-10	4727-9	#6-91393 4727 4512 4729
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>		NA	<1,000 <sup>b</sup>	NA	<1,000
RRPH (Approx.)	400	4,000		<2,000	<4,000	<4,000	<4,000		NA	<4,000	NA	<4,000
VOC 8260												
Benzene	1	1	5	<1	1.2J	<1	<1		<1	<1J	<1J	<1
Carbon Tetrachloride	1	1	5	<1	89J	<1	<1		<1	<1J	<1J	<1
Chloroform	1	1		<1	16J	<1	<1		<1J	<1J	<1J	<1
Toluene	1	1	1,000	<1	9.6J	<1	<1		<1	<1J	<1J	<1
Trichloroethene	1	1	5	<1	62J	<1	<1		<1	<1J	<1J	<1
Xylenes (Total)	2	2	10,000	<2	3.3J	<2	<2		<2	<2J	<2J	<2
SVOC 8270	10	10-11		<10	<10 <sup>c</sup>	<11	<11		NA	NA	NA	<10

☐ CT&E Data.  
☒ F&B Data.  
☒ NA  
☐ J  
☐ b  
☐ c

Result is an estimate.

DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

There were no analytes detected above quantitation limits in this sample, however, some of the surrogate recoveries for this sample were below the validation criteria.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)			Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)					Field Blank		Lab Blank
Parameters	Detect. Limits	Quant. Limits	Action Levels	Cape Lisburne Bkgd. Levels	SW02	SW04	SW05	SW06 & SW07 (Duplicates)		EB02		
ANALYSES	µg/L	µg/L	µg/L	µg/L	4511-2 µg/L	4511-3 µg/L	4511-6 µg/L	4511-7 µg/L	4511-8 µg/L	4511-1 µg/L		4511 µg/L
Laboratory Sample ID Numbers												
Aluminum	17.4	100		<100 (<100)	<100 (<100)	160 (<100)	<100 (<100)	130 (110)	150 (130)	<100 (<100)		<100 (<100)
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100 (<100)
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100 (<100)
Barium	1.2	50	2,000	79-92 (73-89)	77 (71)	210 (180)	860 (720)	460 (440)	490 (470)	<50 (<50)		<50 (<50)
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50 (<50)
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50 (<50)
Calcium	34.5	200		28,000-41,000 (28,000-41,000)	20,000 (19,000)	58,000 (58,000)	150,000 (140,000)	25,000 (27,000)	28,000 (23,000)	<200 (<200)		200 (<200)
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50 (<50)
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<50)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100 (<100)
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)		<50 (<50)
Iron	25	100		<100-190 (<100)	470 (160)	1,600 (330)	4,900 (840)	1,300 (720)	4,000 (2,000)	100 (<100)		<100 (<100)
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)		<100 (<100)

☐ CT&E Data.  
N/A Not available.

**TABLE G-3. LANDFILL AND WASTE ACCUMULATION AREA ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Landfill and Waste Accumulation Area (LF01)										Matrix: Surface Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)				
Parameters	Detect. Limits	Quant. Limits	Action Levels	Cape Lisburne Bkgd. Levels	Environmental Samples				Field Blank		Lab Blank					
					SW02	SW04	SW05	SW06 & SW07 (Duplicates)		EB02						
Laboratory Sample ID Numbers					4511-2	4511-3	4511-6	4511-7	4511-8							
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L							
Magnesium	47.8	200		4,500-9,000 (4,500-8,800)	8,700 (8,400)	14,000 (14,000)	30,000 (28,000)	9,900 (10,000)	10,000 (9,000)	<200 (<200)	200 (<200)					
Manganese	1.24	50		<50 (<50)	<50 (<50)	730 (460)	1,800 (1,100)	90 (<50)	160 (76)	<50 (<50)	<50 (<50)					
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)					
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)					
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)					
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)					
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50J (<50J)	<50 (<50)	<50 (<50)	<50 (<50)	<50J (<50J)	<50 (<50)					
Sodium	27.7	250		4,000-5,600 (3,900-6,000)	38,000 (38,000)	23,000 (23,000)	25,000 (23,000)	19,000 (18,000)	18,000 (17,000)	340 (380)	<250 (<250)					
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)					
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)					
Zinc	8.2	50		<50-260 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)					

☐ CT&E Data.  
☐ N/A Not available.  
☐ J Result is an estimate.

TABLE G-4. WHITE ALICE SITE ANALYTICAL DATA SUMMARY

Installation: Cape Lisburne		Matrix: Soil		Units: mg/kg									
Site: White Alice Site (SS03)		Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blank	Lab Blanks
Parameters						S01	S02	S03	S04-1.75	S05	S06	EB01	#182-9493 #6-9393
Laboratory Sample ID Numbers						1379	1394	1395	1396	1397	1398	1558/1561	#6-9293
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L
DRPH	5-6	50-60	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<50 <sup>b</sup>	<60 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<1,000 <sup>b</sup>	<2,000
RRPH (Approx.)	10-20	100-200	2,000 <sup>a</sup>	<120 <300	<110	<120	<110	<200	<100	<100	<110	<2,000	<4,000
Pesticides	0.005-0.05	0.05-0.5	0.04	<0.01J-0.07R	NA	NA	NA	NA	NA	NA	<0.05 <0.5	<0.2J <10J	<0.2 <10 <0.01 <0.5
PCBs													
Aroclor 1260	0.001	0.1	10	<0.02-20JN	<0.1	0.8JN	5.5JN	20JN	1JN	2JN	<2J	<10	<0.1 <0.5

□

CT&amp;E Data.

■

F&amp;B Data.

NA

Not analyzed.

J

Result is an estimate.

N

The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

R

Result has been rejected.

a

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b

DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

TABLE G-4. WHITE ALICE SITE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: White Alice Site (SS03)			Matrix: Soil Units: mg/kg										
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blank	Lab Blanks	
					2S07-2 & 2S13-2 (Replicates)	2S08	2S09-2.5	2S10-1.5	2S11-2.5				
Laboratory Sample ID Numbers					1908	1913	1909	1910	1911	1912	1924	#6-91393	#6-91393
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	mg/kg
DRPH	5-10	50-100	500 <sup>a</sup>	<50 <sup>b</sup> <150 <sup>b</sup>	<100 <sup>b</sup>	<50J <sup>b</sup>	380J <sup>b</sup>	<50J <sup>b</sup>	<50J <sup>b</sup>	<50J <sup>b</sup>	<1,000 <sup>b</sup>	<1,000	<50J
RRPH (Approx.)	10-20	100-200	2,000 <sup>a</sup>	<120 <300	<100	<200	<100	<200	<100	<100	<4,000	<4,000	<100
PCBs													
Aroclor 1260	0.01	0.1	10	<0.02-20JN	0.5J	1.8J	<0.1	<0.1	<0.1	<0.1	<2	<10	<0.1

☐ CT&E Data.  
☒ F&B Data.  
☒ NA  
☐ Not analyzed.  
☐ Result is an estimate.  
☐ The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".  
☐ The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.  
☐ DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.



TABLE G-4. WHITE ALICE SITE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: White Alice Site (SS03)		Matrix: Soil Units: mg/kg								
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blank	Lab Blanks	
					3S19 & 3S23 (Replicates)	3S22-0.5	3S33	3EB01		
Laboratory Sample ID Numbers					4608-11	4608-12	4762-5	4608-5	4608 4762	4608
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	mg/kg	µg/L
PCBs										
Aroclor 1260	0.020	0.020	10	<0.02-20.1N	300	233	6,290	26.8	<1.00	<0.03
										<1

□ CT&E Data.

■ F&B Data.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Soil Units: mg/kg										Field Blanks				Lab Blanks	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples								AB01	EB03	TB03		
Laboratory Sample ID Numbers					S01-1	S02-2.5	S03-5.5	S04-2	S05-3	S06-3			4512-3	1625/1628	1626	4512 #6-9393 #3&4-9493	#6-9393 #3&4-9493
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg			μg/L	μg/L	μg/L	μg/L	mg/kg
DRPH	5-6	50-60	500 <sup>a</sup>	<60 <sup>b</sup> , 190 <sup>b</sup>	63,000 <sup>b</sup>	2,300 <sup>b</sup>	4,300 <sup>b</sup>	<50 <sup>b</sup>	<60 <sup>b</sup>	<50 <sup>b</sup>			NA	<1,000 <sup>b</sup>	NA	<2,000	<50
GRPH	0.1-0.9	1-9	100	<2 <sup>b</sup> , <6 <sup>b</sup>	43 <sup>b</sup>	13 <sup>b</sup>	25 <sup>b</sup>	25 <sup>b</sup>	<8 <sup>b</sup>	<1 <sup>b</sup>			NA	<50 <sup>b</sup>	<50 <sup>b</sup>	<50	<1-<20
RRPH (Approx.)	10-200	100-2,000	2,000 <sup>a</sup>	<120-<300	<2,000	<100	<100	<100	<120	<100			NA	<2,000	NA	<4,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.3	<3.8	<2.5	<0.28	<4.12	<1.04	<1.0							
Benzene	0.002-0.07	0.02-0.7	0.5	<0.02-<0.08	<0.4	<0.7	<0.02	<0.02	<0.02	<0.2			<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2
Toluene	0.002-0.07	0.02-0.7		<0.02-<0.08	<0.4	<0.7	<0.02	<0.5	<0.2	<0.2			<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2
Ethylbenzene	0.002-0.2	0.02-2		<0.02-<0.08	<2	<0.8	<0.04	<0.8	<0.02	<0.2			<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2
Xylenes (Total)	<0.02-<0.3	<0.2-<3		<0.04-<0.12	<0.8	<0.5	<0.2	<3	<0.8	<0.4			<2 <sup>c</sup>	<2	<2	<2	<0.04-<0.4

□ CT&E Data.

■ F&B Data.

■ NA

■ Not analyzed.

Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)													Matrix: Soil Units: mg/kg												
Parameters		Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks											
						S07-4	S08-1.5	S09-3	S10-1.5	S11-2	AB01	EB03	TB03												
Laboratory Sample ID Numbers						1616	1618	1620	1622	1624	4512-13	1625/1628	1626	4512 #6-9393 #182-9493	#6-9393 #384-9493										
ANALYSES		mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg										
DRPH		5	50	500 <sup>a</sup>	<60 <sup>b</sup> <130 <sup>b</sup>	3,900 <sup>b</sup>	<50 <sup>b</sup>	180 <sup>b</sup>	20,000 <sup>b</sup>	800 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<2,000	<50										
GRPH		0.1	1	100	<2.0 <sup>b</sup> <6.0 <sup>b</sup>	<1.0 <sup>b</sup>	<1.0 <sup>b</sup>	11.0 <sup>b</sup>	111.0 <sup>b</sup>	48.0 <sup>b</sup>	NA	<30.0 <sup>b</sup>	<30.0 <sup>b</sup>	<50	<1-<20										
RRPH (Approx.)		10-16	100-160	2,000 <sup>a</sup>	<120-<300	<100	<100	<100	<160	<100	NA	<2,000	NA	<4,000	<100										
BTEX (8020/8020 Mod.)				10 Total BTEX	<0.1-<0.3	0.18J	0.03	1.6J	18.4J	5.5J															
Benzene		0.002-0.04	0.02-0.4	0.5	<0.02-<0.06	<0.02	<0.02	<0.02	1.4J	<0.02	<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2										
Toluene		0.002-0.04	0.02-0.4		<0.02-<0.06	0.03	0.03	0.4	<0.3	<0.02	<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2										
Ethylbenzene		0.002-0.04	0.02-0.4		<0.02-<0.06	0.05J	<0.03	0.6	7	3.2J	<1 <sup>c</sup>	<1	<1	<1	<0.02-<0.2										
Xylenes (Total)		0.004-0.08	0.04-0.8		<0.04-0.12	0.1J	<0.06	0.6J	10J	2.3J	<2 <sup>c</sup>	<2	<2	<2	<0.04-<0.4										

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX was determined by 8260 method analysis.

**TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)					Matrix: Soil Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks	
					2S12-4.5 & 2S17-4.5 (Replicates)	2S13-1.5	2S14-5	2S15	2S16-3	AB01	2EB04	2TB04		
Laboratory Sample ID Numbers					1929 4728-15	1934 4728-20	1930 4728-18	1931 4728-17	1932 4728-18	1933 4728-18	4512-3	1924 4727-10	4727-9	#6-91393 4727 4512
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	mg/kg
DRPH	6-7	60-70	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<60 <sup>a</sup>	<60 <sup>b</sup>	<60 <sup>a</sup>	<70J <sup>b</sup>	<70J <sup>b</sup>	<70J <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<1,000
RRPH (Approx.)	12-14	120-140	2,000 <sup>a</sup>	<120 <300	<120	<120	<120	<140	<140	<140	NA	<4,000	NA	<4,000
VOC 8260														
1,4-Dichlorobenzene	0.020	0.025		<0.025 <0.160	<0.025J	<0.025	<0.025	<0.025	0.095	<0.025	<1	<1J	<1J	<0.025
Ethylbenzene	0.020	0.025	0.025	<0.025 <0.160	0.030J	<0.025	<0.025	<0.025	<0.025	<0.025	<1	<1J	<1J	<0.025
Naphthalene	0.020	0.025		<0.025 <0.160	<0.025J	0.068	0.035	0.027	<0.025	0.038	<1	<1J	<1J	<0.025
Toluene	0.020	0.025		<0.025 <0.160	0.045J	0.029	0.033	<0.025	<0.025	0.036	<1	<1J	<1J	<0.025
Trichloroethene	0.020	0.025		<0.025 <0.160	<0.025J	<0.025	<0.025	<0.025	0.059	<0.025	<1	<1J	<1J	<0.025
1,2,4-Trimethylbenzene	0.020	0.025		<0.025 <0.160	0.087J	0.108	0.078	<0.025	0.030	0.051	<1	<1J	<1J	<0.025
1,3,5-Trimethylbenzene	0.020	0.025		<0.025 <0.160	0.048J	0.059	<0.025	<0.025	<0.025	0.037	<1	<1J	<1J	<0.025
Xylenes (Total)	0.040	0.050		<0.050 <0.320	0.155J	0.116	0.055 <sup>d</sup>	<0.500	0.043 <sup>d</sup>	0.132	<2	<2J	<2J	<0.050

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

d Result is indicative of p&m-xylenes only.

TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Soil Units: mg/kg		Environmental Samples				Field Blank		Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	2S18-3	2S19	2S20		2EB04	#6-91393	#6-91393
Laboratory Sample ID Numbers					1935	1936	1937		1924		
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg		µg/L		mg/kg
DRPH	7.0	70	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<70 <sup>b</sup>	1,100J <sup>b</sup>	490J <sup>b</sup>		<1,000 <sup>b</sup>	<1,000	<50J
RRPH (Approx.)	14-20	140-200	2,000 <sup>a</sup>	<120 <300	<140	1,200	<200		<4,000	<4,000	<100

F&B Data.

Result is an estimate.

The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

J  
a  
b

TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)			Matrix: Soil Units: mg/kg			Environmental Samples										Field Blanks			Lab Blanks	
Parameters	Detect Limits	Quant. Limits	Action Levels	Bkgd. Levels	3S21-4	3S22-5.5	3S23 & 3S25 1' to 3' Composite (Replicates)	3S24 1' to 2' Composite	LF01 AB01	ST07 3TB01	3EB03									
Laboratory Sample ID Numbers					4639-5	4639-6	4639-7	4639-8	4762-1	4639-3	4639-4	4639	4762	4639	4762	4639	4762			
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	mg/kg	mg/kg			
DRPH	4.00		500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>c</sup>	1,190	125	151	2,040	NA	NA	<100	<100	<100	<100	<100	<4.00	<4.00			
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.3	0.467	<0.125	0.096	0.493	5.147											
Benzene	0.020	0.025-0.030	0.5	<0.02-<0.06	<0.030	<0.025	<0.025	<0.025	<1 <sup>c</sup>	<1	<1	<1	<1	<1	<1	<0.020	<0.020			
Toluene	0.020	0.025-0.030		<0.02-<0.06	<0.030	<0.025	<0.025	0.097	<1 <sup>c</sup>	<1	<1	<1	<1	<1	<1	<0.020	<0.020			
Ethylbenzene	0.020	0.025-0.030		<0.02-<0.06	0.092	<0.025	<0.025	0.139	<1 <sup>c</sup>	<1	<1	<1	<1	<1	<1	<0.020	<0.020			
Xylenes (Total)	0.040	0.050-0.060		<0.04-<0.12	0.375	<0.050	0.096	0.257	<2 <sup>c</sup>	<2	<2	<2	<2	<2	<2	<0.040	<0.040			

□ CT&E Data.

■ F&B Data.

Not analyzed.

The action level for DRPH is based on conversations with ADEC; a final action level has not yet been determined.

DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

□

■

NA

a

b

c

**TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)					Matrix: Soil Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blank	
					3S28-5	3S27-2.5	3S28-6.5	3S29-3.5	3S30-6.5	3S31-5	LF01 AB01	ST07 3TB01	3EB03	
Laboratory Sample ID Numbers					4639-10	4639-11	4639-12	4639-13	4639-14	4639-15	4762-1	4639-3	4639-4	4639
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	mg/kg
DRPH	4.00	4.00	500 <sup>a</sup>	< 60 <sup>b</sup> < 150 <sup>c</sup>	11,800	228	8,180	1,790	518	3,130	NA	NA	<100	<4.0
BTEX (8020/8020 Mod.)			10 Total BTEX	< 0.1 < 0.3	7,811	< 0.100	< 0.125	< 0.100	< 0.125	6,813				
Benzene	0.020	0.020-0.025	0.5	< 0.02 < 0.06	< 0.025	< 0.020	< 0.025	< 0.020	< 0.025	< 0.025	< 1 <sup>c</sup>	< 1	< 1	< 0.5 < 1
Toluene	0.020	0.020-0.025		< 0.02 < 0.06	0.949	< 0.020	< 0.025	< 0.020	< 0.025	0.113	< 1 <sup>c</sup>	< 1	< 1	< 0.5 < 1
Ethylbenzene	0.020	0.020-0.025		< 0.02 < 0.06	4.53	< 0.020	< 0.025	< 0.020	< 0.025	2.66	< 1 <sup>c</sup>	< 1	< 1	< 0.5 < 1
Xylenes (Total)	0.040	0.040-0.050		< 0.04 < 0.12	4.23	< 0.040	< 0.050	< 0.040	< 0.050	4.04	< 2 <sup>c</sup>	< 2	< 2	< 1 < 2

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

The action level for DRPH is based on conversions with ADEC; a final action level has not yet been determined.

DRPH concentrations reported for these samples are equivalent to diesel (DRO) as defined by ADEC.

BTEX determined by 8260 method analysis.

□

■

NA

a

b

c

TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Sediment Units: mg/kg											
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks
					SD01 & SD06 (Replicates)	SD02	SD03	SD04	SD05	AB01	EB03	TB03	
Laboratory Sample ID Numbers					1644	1654	1646	1648	1650	4512-3	1625/1628	1626	#6-9393 #6-91093 #38-4-9493
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	μg/L
DRPH	6	60	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	1,400 <sup>b</sup>	2,400 <sup>b</sup>	<60 <sup>b</sup>	<60 <sup>b</sup>	850 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<2,000
GRPH	1.3-2.0	13-20	100	<2.0 <sup>b</sup> <6.0 <sup>b</sup>	138 <sup>b</sup>	143 <sup>b</sup>	150 <sup>b</sup>	<13 <sup>b</sup>	20 <sup>b</sup>	NA	<50 <sup>b</sup>	<50 <sup>b</sup>	<50
RRPH (Approx.)	12-20	120-200	2,000 <sup>a</sup>	<120 <300	<120	<130	<120	<200	<200	NA	<2,000	NA	<1 <20
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1 <0.3	4.7 J	2.5 J	<1.04	<2.64 J	2.97 J				<4,000
Benzene	0.002-0.004	0.02-0.04	0.5	<0.02 <0.06	<0.02	0.3	<0.02	<0.02	<0.04	<1 <sup>c</sup>	<1	<1	<0.02 <0.2
Toluene	0.002-0.004	0.02-0.04		<0.02 <0.06	<0.02	0.3	<0.02	<0.04	0.07	<1 <sup>c</sup>	<1	<1	<0.02 <0.2
Ethylbenzene	0.002-0.08	0.02-0.8		<0.02 <0.06	0.9	0.5	<0.8	<0.4	0.9	<1 <sup>c</sup>	<1	<1	<0.02 <0.2
Xylenes (Total)	0.004-0.18	0.04-1.8		<0.04 <0.12	3.6 J	1.4 J	<0.4	<0.9	2 J	<2 <sup>c</sup>	<2	<2	<0.04 <0.4

□ CT&amp;E Data.

■ F&amp;B Data.

NA Not analyzed.

J Result is an estimate.

a The actions level for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTEX determined by 8260 method analysis.



TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Sediment Units: mg/kg		Environmental Samples				Field Blanks			Lab Blanks	
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	SD01 & SD06 (Replicates)		SD04	AB01	EB03	TB03		
Laboratory Sample ID Numbers					1644 4614-8	1654 4614-10	1650 4614-9	4512-3	1625 4614-2	4614-1	4512/4614 #6-9393	4614 #6-9393
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
VOC 8260												
sec-Butylbenzene	0.020	0.025-0.050		<0.025-<0.160	0.370J	<0.025J	<0.050	<1	<1	<1	<1	<0.020
Ethylbenzene	0.020	0.025-0.050		<0.025-<0.160	0.242J	0.134J	0.072	<1	<1	<1	<1	<0.020
Isopropylbenzene	0.020	0.025-0.050		<0.025-<0.160	0.210J	0.085J	<0.050	<1	<1	<1	<1	<0.020
p-Isopropyltoluene	0.020	0.025-0.050		<0.025-<0.160	0.762J	0.435J	0.157	<1	<1	<1	<1	<0.020
n-Propylbenzene	0.020	0.025-0.050		<0.025-<0.160	0.366J	0.149J	0.053	<1	<1	<1	<1	<0.020
Toluene	0.020	0.025-0.050		<0.025-<0.160	0.074J	0.037J	0.047	<1	<1	<1	<1	<0.020
Trichloroethene	0.020	0.025-0.050		<0.025-<0.160	<0.025	<0.025	0.426	<1	<1	<1	<1	<0.020
1,2,4-Trimethylbenzene	0.020	0.025-0.050		<0.025-<0.160	2.81	1.97	0.953	<1	<1	<1	<1	<0.020
1,3,5-Trimethylbenzene	0.020	0.025-0.050		<0.025-<0.160	1.76	1.27	0.546	<1	<1	<1	<1	<0.020
Xylenes (Total)	0.040	0.050-0.100		<0.050-<0.320	0.776J	0.548J	0.295	<2	<2	<2	<2	<0.040
SVOC 8270												
di-n-Butylphthalate	0.200	0.311-8.20	8.000	1.61U-20.4JB	NA	6.74U	31.5JB	NA	NA	NA	NA	0.800
2-Methyl-naphthalene	0.200	0.311-8.20		<0.250-<4.23	NA	5.12J	2.29J	NA	NA	NA	NA	<0.200
4-Methylphenol	0.200	0.311-8.20		<0.250-<4.23	NA	<0.311J	7.32J	NA	NA	NA	NA	<0.200
Naphthalene	0.200	0.311-8.20		<0.250-<4.23	NA	4.18J	<8.20	NA	NA	NA	NA	<0.200
Pesticides	0.001	0.01		<0.01-0.07	<0.01	<0.01	NA	NA	<0.2-<10	NA	<0.2-<10	<0.01

☐ CT&E Data.  
☒ F&B Data.  
☒ NA  
 Not analyzed.  
 B The analyte was detected in the associated blank.  
 J Result is an estimate.  
 U Compound is not presented above the concentration listed.

**TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Sediment Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
Laboratory Sample ID Numbers					2SD07	2SD08		AB01	2EB04	2TB04	
					1938 4738-13	1939 4728-14		4512-3	1924 4727-10	4727-9	#6-91393 4727 4512
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg		µg/L	µg/L	µg/L	mg/kg
DRPH	10	100	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<100J <sup>b</sup>	<100J <sup>b</sup>		NA	<1,000 <sup>b</sup>	NA	<50J
RRPH (Approx.)	20	200	2,000 <sup>a</sup>	<120 <300	<200	<200		NA	<4,000	NA	<100
VOC 8260											
p-Isopropyltoluene	0.020	0.140-0.200		<0.025-<0.160	<0.200J	0.145		<1	<1J	<1J	<0.200
Napthalene	0.020	0.140-0.200		<0.025-<0.160	<0.200J	0.380		<1	<1J	<1J	<0.200
1,2,4-Trimethylbenzene	0.020	0.140-0.200		<0.025-<0.160	<0.200J	0.185		<1	<1J	<1J	<0.200

☐ CT&E Data.  
☒ F&B Data.  
☒ NA  
☒ J  
☒ a  
☒ b

Not analyzed  
 Result is an estimate.  
 The action levels for DRPH and RRRPH are based on conversations with ADEC; final action levels have not yet been determined.  
 DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Surface Water Units: µg/L		Environmental Samples			Field Blanks			Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	SW01 & SW03 (Duplicates)		AB01	EB03	TB03	
Laboratory Sample ID Numbers					1631/1632 4614-3	1639/1640 4614-5	4512-3	1625/1628	1626	#6-9393 #1&2-9493 4614
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<2,000
GRPH	5	50		<50 <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	NA	<50 <sup>b</sup>	<50 <sup>b</sup>	<50
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	NA	<2,000 <sup>c</sup>	NA	<4,000
BTX (8020/8020 Mod.)										
Benzene	0.1	1	5	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1	<1	<1
Toluene	0.1	1	1,000	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1	<1	<1
Ethylbenzene	0.1	1	700	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup>	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2 <sup>c</sup>	<2 <sup>c</sup>	<2 <sup>c</sup>	<2 <sup>c</sup>	<2	<2	<2
VOC 8260										
Naphthalene	1	1		<1	1.5	1.7	<1	<1	<1	<1
1,2,4-Trimethylbenzene	1	1		<1	1.1	1.2	<1	<1	<1	<1
SVOC 8270	10	11		<10	<11	<11	NA	NA	NA	<10

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

c BTX determined by 8260 method analysis.

**TABLE G-5. SPILL/LEAK #3 ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Spill/Leak #3 (ST07)		Matrix: Surface Water Units: µg/L									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks			Lab Blanks
					SW01 & SW03 (Duplicates)			AB01	EB03	TB03	
Laboratory Sample ID Numbers					1631/1632 4614-3	1639/1640 4614-5		4512-3	1625/1628	1626	4614
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		µg/L	µg/L	µg/L	µg/L
Pesticides	0.02-1.0	0.2-10		<0.23-10J	<0.2-10	<0.2-10		NA	<0.2-10	NA	NA
TOC	5,000	5,000		<5,000-15,600	12,000	12,600		NA	NA	NA	<5,000
TSS	100	200		2,500-3,000	9,000	10,000		NA	NA	NA	<200
TDS	10,000	10,000		203,000- 245,000	369,000	376,000		NA	NA	NA	<10,000

☐ CT&E Data.  
☒ F&B Data.  
☒ Not analyzed.  
☒ Result is an estimate.  
 J

TABLE G-6. UPPER CAMP TRANSFORMER BUILDING ANALYTICAL DATA SUMMARY

Installation: Cape Lisburne		Matrix: Soil										
Site: Upper Camp Transformer Building (SS08)		Units: mg/kg										
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blank	Lab Blanks	
					S01	S02	S03 & S07 (Replicates)	S04	S05-2			
Laboratory Sample ID Numbers					1399	1400	1401	1404 4477-4	1402	1403	1558/1561	#1 & 2-9493 #6-9393
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L
DRPH	5.0	50	500 <sup>a</sup>	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	750 <sup>b</sup>	19,500 <sup>b</sup>	51,000 <sup>b</sup>	50 <sup>b</sup>	330 <sup>b</sup>	<1,000 <sup>b</sup>	<2,000
RRPH (Approx.)	N/A	N/A	2,000 <sup>a</sup>	2,000 <sup>a</sup>	<120 <300	460	6,500	29,000	250	430	<2,000	<4,000
PCBs												
Aroclor 1260	0.01-0.1	0.1-1	10	<0.02-20 <sup>HN</sup>	9 <sup>HN</sup>	300 <sup>HN</sup>	130 <sup>JN</sup>	8 <sup>JN</sup>	0.9 <sup>JN</sup>	<0.1	<2 <sup>J</sup>	<0.1
TOC				25,700-77,900	NA	NA	NA	67,300	NA	NA	NA	NA

TABLE G-6. UPPER CAMP TRANSFORMER BUILDING ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne		Matrix: Soil									
Site: Upper Camp Transformer Building (SS08)		Units: mg/kg									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples			Field Blanks		Lab Blanks	
Laboratory Sample ID Numbers					2S08	2S09	3S12	2EB04	3EB01	#6-91393 4608	#6-91393 4608
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L	μg/L	mg/kg
DRPH	5	50	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	<100 <sup>b</sup>	<50 <sup>b</sup>	NA	<1,000 <sup>b</sup>	NA	<1,000	<50J
RRPH (Approx.)	10	100	2,000 <sup>a</sup>	<120 <300	<100	<100	NA	<4,000	NA	<4,000	<100
PCBs											
Aroclor 1254	0.01-0.020	0.020-0.1	10	<0.02 <0.3	<0.1	<0.1	29.1	<2	<1.00	<10	<0.1

☐ GT&E Data.  
☒ F&B Data.  
☒ Not analyzed.  
☒ NA  
 J Result is an estimate.  
 a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.  
 b DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

**TABLE G-7. LOWER CAMP TRANSFORMER BUILDINGS ANALYTICAL DATA SUMMARY**

Installation: Cape Lisburne		Matrix: Soil											
Site: Lower Camp Transformer Buildings (SS09)		Units: mg/kg											
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples							Field Blank	Lab Blanks
					S01	S03	S04	S05	S06	S07	EB01		
Laboratory Sample ID Numbers					1383	1384	1385	1386	1387	1388		1558 1561	#6-9393 #1&2-9493 #6-9593
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L
DRPH	5	50	500 <sup>a</sup>	<60 <sup>b</sup> , <150 <sup>b</sup>	760J <sup>b</sup>	13,000J <sup>b</sup>	<50 <sup>b</sup>	<50 <sup>b</sup>	<50J <sup>b</sup>	<50 <sup>b</sup>	<1,000J <sup>b</sup>	<2,000	<50
RRPH (Approx.)	10	100	2,000 <sup>a</sup>	<120-<300	4,500	4,500	<100	<100	<100	<100	<2,000	<4,000	<100
Pesticides													
Endosulfan 1	0.001	0.01	4	<0.01J	NA	NA	NA	NA	0.03R	NA	<0.2J	<0.2	<0.01
Endrin Aldehyde	0.001	0.01		<0.01J	NA	NA	NA	NA	0.02R	NA	<0.2J	<0.2	<0.01
Endosulfan Sulfate	0.001	0.01		<0.01J	NA	NA	NA	NA	0.02R	NA	<0.2J	<0.2	<0.01
PCBs													
Aroclor 1260	0.01-0.1	0.1-1	10	<0.02-20JN	340JN <sup>d</sup>	14JN <sup>d</sup>	2JN	0.1	1.4JN	<0.1	<2J	<10	<0.1

☐ CT&E Data.  
☒ F&B Data.  
☒ NA Not analyzed.  
 J Result is an estimate.  
 N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".  
 R Result has been rejected.  
 a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.  
 b DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.  
 d The laboratory reported the results were outside of the calibration range.

TABLE G-7. LOWER CAMP TRANSFORMER BUILDINGS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Lower Camp Transformer Buildings (SS09)					Matrix: Soil Units: mg/kg						
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blank	Lab Blanks	
					S08 & S12 (Replicates)	S09-2	S10	S11			
Laboratory Sample ID Numbers					1389	1393	1390	1391	1392	1558/1561	#6-9393 #1&2-9493
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	μg/L	μg/L
DRPH	5	50	500 <sup>a</sup>	<60 <sup>b</sup> <150 <sup>b</sup>	30,000J <sup>b</sup>	25,000J <sup>b</sup>	4,900J <sup>b</sup>	<50J <sup>b</sup>	<50 <sup>b</sup>	<1,000J <sup>b</sup>	<2,000
RRPH (Approx.)	10	100	2,000 <sup>a</sup>	<120 <300	12,000	13,000	2,700	230	<100	<2,000	<4,000
PCBs											
Aroclor 1260	0.01-0.1	0.1-1	10	<0.02-20JN	380JN <sup>d</sup>	550JN <sup>d</sup>	350JN <sup>d</sup>	22JN	<0.1	<2J	<10
											<0.1-<0.5

☐ CT&E Data.  
☒ F&B Data.  
☒ Not analyzed.  
 Result is an estimate.  
 The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".  
 The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.  
 DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.  
 The laboratory reported the results were outside of the calibration range.



TABLE G-7. LOWER CAMP TRANSFORMER BUILDINGS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne		Matrix: Soil			Units: mg/kg		
Site: Lower Camp Transformer Buildings (SS09)		Environmental Samples			Field Blank		
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	3S13 & 3S14 (Replicates)		
					3S15	3EB01	Lab Blanks
Laboratory Sample ID Numbers					4608-2	4608-1	4608
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	mg/kg
PCBs							
Aroclor 1254	0.020	0.020	10	<0.02	1,010	<0.020	<0.03
Aroclor 1260	0.020	0.020	10	<0.02	<0.020	<1.00	<0.03

☐ CT&E Data.  
☒ F&B Data.  
 Result is an estimate.  
 The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".

**TABLE G-8. WATER GALLERY ANALYTICAL DATA SUMMARY**

Installation: Cape Lisburne Site: Water Gallery (AOC3)			Matrix: Surface/Ground Water Units: µg/L									
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks
					SW01	GW01	GW02	GW03	AB01	EB01	TB01	
Laboratory Sample ID Numbers					1380 4477-3 4476-4	1570 4481-1 4477-7 4476-8	1571	1572	4512-3	1558/1561 4476-5	1552 4476-1	#1&2-9493 #6-9393 4476 4481 4477
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	20	200		<1,000 <sup>b</sup>	<1,000 <sup>b</sup>	NA	NA	NA	NA	<1,000J <sup>b</sup>	NA	NA
RRPH (Approx.)	200	2,000		<2,000	<2,000	NA	NA	NA	NA	<2,000	NA	NA
VOC 8240	1	1-10		<1 <sup>c</sup>	<1-<10	<1-<10	<1	<1-<10	<1 <sup>c</sup> -13J <sup>c</sup>	<1 <sup>c</sup>	<1 <sup>c</sup> -1.7 <sup>c</sup>	<1
SVOC 8270	10	10-11		<10	<10	<11	<10	<10	NA	<10	NA	<10
Pesticides	0.02-1.0	0.2-10		<0.2J-<10J	<0.2-<10	<0.2J-<10J	<0.2-<10	<0.2J-<10J	NA	<0.2J-<10J	NA	NA
PCBs	0.2	2	0.5	<2J	<2J	<2J	NA	<2J	NA	<2J	NA	NA
TOC	5,000	5,000		<5,000-15,600	<5,000	<5,000	<5,000	<5,000	NA	NA	NA	NA
TSS	100	200		<2,500-3,000	2,500	4,500	4,000	4,000	NA	NA	NA	<200
TDS	10,000	10,000		203,000-245,000	235,000	183,000	176,000	180,000	NA	NA	NA	<10,000

□ CT&E Data.

■ F&B Data.

NA Not analyzed.

J Result is an estimate.

b DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

c Result determined by 8260 method analysis.

TABLE G-8. WATER GALLERY ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Cape Lisburne Site: Water Gallery (AOC3)		Matrix: Ground Water Units: µg/L		Environmental Sample				Field Blanks			Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	2GW04			AB01	2EB04	2TB04	
Laboratory Sample ID Numbers					1928 4727-1			4512-3	1924 4727-10	4727-9	#6-91393 4727
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L	µg/L
VOC 8260	1	1		<1	<1J			<1-13J	<1-3.5J	<1-7.0J	<1
SVOC 8270	10	10		<10	<10			NA	NA	NA	<10
Pesticides	0.02-10	0.2-10		<0.2J-<10J	<0.2J-<10J			NA	NA	NA	NA
PCBs	0.2	2	0.5	<2J	<2			NA	NA	NA	<0.1

☐ CT&E Data.  
☒ F&B Data.  
☒ Not analyzed.  
 J Result is an estimate.

**TABLE G-8. WATER GALLERY ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Water Gallery (AOC3)		Matrix: Surface/Ground Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)								Field Blanks		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	SW01	GW01	GW02	GW03	2GW04	EB01	EB02			
Laboratory Sample ID Numbers					4477-5	4481-1	4481-2 4476-9	4476-10	4727-1	4476-5	4511-2			4476 4477/4481 4511/4727
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
Aluminum	17.4	100		<100 (<100)	<100J (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100J (<100)	<100	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)
Barium	1.2	50	2,000	79-92 (73-89)	100 (100)	87 (85)	130 (120)	92 (90)	73	<50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)
Calcium	34.5	200		28,000-41,000 (28,000-41,000)	41,000 (41,000)	41,000 (41,000)	41,000 (41,000)	43,000 (42,000)	32,000	210	<200 (<200)	<200 (<200)	<200 (<200)	<200 (<200)
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)
Cobalt	N/A	50-100		<100 (<100)	<100 (<100)	<100 (<100)	<50 (<100)	<100 (<100)	<100	<100	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)
Iron	25.0	100		<100 (<100)	120 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100J	110	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)

☐ CT&E Data.  
☐ N/A Not available.  
☐ J Result is an estimate.

**TABLE G-8. WATER GALLERY ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Water Gallery (AOC3)			Matrix: Surface/Ground Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)							Field Blanks		Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	SW01	GW01	GW02	GW03	2GW04	EB01	EB02			
Laboratory Sample ID Numbers					4477-5	4481-1	4481-2 4476-9	4476-10	4727-1	4476-5	4511-2	4476 4477/4481 4511/4727		
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L		
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100	<100	<100	<100 (<100)		<100 (<100)
Magnesium	47.8	200		4,500-9,000 (4,500-8,800)	8,900 (8,900)	8,400 (8,400)	8,700 (8,400)	9,300 (8,800)	7,000	<200	<200	<200 (<200)		<200 (<200)
Manganese	1.24	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50	<50 (<50)		<50 (<50)
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50	<50 (<50)		<50 (<50)
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50	<50 (<50)		<50 (<50)
Potassium	1,154.3	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000	<5,000	<5,000	<5,000 (<5,000)		<5,000 (<5,000)
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)	<100	<100	<100	<100 (<100)		<100 (<100)
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50J	<50 (<50J)		<50 (<50)
Sodium	27.7	250		4,000-5,600 (3,900-6,000)	4,400 (4,600)	5,100 (5,300)	5,000 (5,300)	5,300 (4,600)	4,800	360	340 (380)	<250 (<250)		<250 (<250)
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)	<5	<5	<5	<5 (<5)		<5 (<5)
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50	<50	<50	<50 (<50)		<50 (<50)

☐ CT&E Data.  
☐ N/A Not available.  
☐ J Result is an estimate.

**TABLE G-8. WATER GALLERY ANALYTICAL DATA SUMMARY (CONTINUED)**

Installation: Cape Lisburne Site: Water Gallery (AOC3)		Matrix: Surface/Ground Water Units: µg/L		METALS ANALYSES: TOTAL (DISSOLVED)								Lab Blanks
Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			
					SW01	GW01	GW02	GW03	2GW04	EB01	EB02	
Laboratory Sample ID Numbers					4477-5	4481-1 4476-9	4481-2 4476-9	4476-10	4727-1	4476-5	4511-2	4476 4477/4481 4511/4727
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
Zinc	8.2	50		<20-260 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)	190	<50 (<50)	<50 (<50)	<50 (<50)

☐ CT&E Data.  
☐ N/A Not available.